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Traffic in Persons and Prostitution*

A study on traffic in persons and prostitution has now been published in booklet form by the United Nations. The report, originally prepared by the UN Secretariat for the twelfth session (April-May 1959) of the Social Commission of the Economic and Social Council has, in its printed form, been brought up to date as of 1 August 1959.

The study contains a programme of action which is designed to strengthen the provisions already embodied in the Convention for the Suppression of the Traffic in Persons and of the Exploitation of Others, adopted by the UN General Assembly in 1949. The Economic and Social Council, in July 1959, recommended that governments bring the programme of action to the attention of the governmental agencies concerned and, when appropriate, the non-governmental organizations interested in this field.

The 1949 UN Convention consolidates earlier international instruments on the subject and embodies the so-called 'abolitionist' policy. This policy comprises the abolition of any form of the regulation of prostitution, the repression of third-party profiteers, the prevention of prostitution and the rehabilitation of its victims.

In advocating the abolitionist system, the UN experts in their *Report* oppose the 'prohibitionist' system which makes prostitution *per se* a criminal offence. The latter method, it is said, 'creates among persons engaged in prostitution a collective as well as an individual antagonistic

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*Study on Traffic in Persons and Prostitution: Suppression of the Traffic in Persons and of the Exploitation of the Prostitution of Others.** A Report Published by the United Nations, Department of Economic and Social Affairs. (1959. Pp. 57. 3s. 6d.).

International Documents Service, Columbia University Press, 2960 Broadway, New York 27, N.Y.

Pretoria: Van Schaik's Bookstore (Pty.) Ltd., P.O. Box 724.

attitude which hampers their rehabilitation. It also leads generally to an organized clandestine prostitution which militates against the effectiveness of a programme of action.'

However, the adoption of the abolitionist system, the *Report* states, is insufficient by itself and should be accompanied by measures aimed at the prevention of prostitution, the rehabilitation of persons engaged in prostitution, the repression of the traffic in persons and of the exploitation of the prostitution of others, and the prevention of venereal disease. It is emphasized that the corresponding programme, policies and services must be well co-ordinated.

In part of the *Report* the authors review international action already taken in this field, analyse the extent and nature of the problem, and deal with such aspects as the abolition of the regulation of prostitution, the legal status of prostitution, the prevention of venereal disease, the prevention of prostitution, legal provisions proscribing the traffic in persons and the exploitation of the prostitution of others, and the re-education of persons engaged in prostitution.

A separate Chapter then recommends a series of measures to counteract and prevent prostitution—including those of a general social and economic character as well as specific services and practices.

The full text of the Consolidated Convention and a Table showing signatures and ratifications or accessions are also given in the UN booklet.

Second South African Medico-Legal Congress

This Congress will be held in Durban from 3-7 July 1960. Those interested in attending should communicate without delay with the Honorary Organizing Secretary, Prof. I. Gordon, Dean of the Faculty of Medicine, University of Natal, Durban.

Second International Meeting on Forensic Pathology

This conference will be held in New York City from 18–22 September 1960. All inquiries in connexion with this meeting should be addressed to:

Dr. Milton Helpern, 55 East End Avenue, New York 28, N.Y., U.S.A.

Abstracts

The Effect of Age on Taste Sensitivity

Canadian authors have studied the question of how sensitivity to the four primary taste qualities is modified by ageing. A hundred test subjects of all ages between 15 and 89 years were given solutions of sucrose, sodium chloride, hydrochloride acid, and quinine sulphate in various concentrations. The subjects each received two beakers, one containing the test solution and the other distilled water; they were allowed to sip the two liquids as often as they wanted until they had made up their mind. The first objective was to find out when the test solution tasted different from the water (difference threshold). By increasing the concentration further, the 'taste threshold' was ascertained, i.e. the point at which the subject reported a definite taste quality—e.g. 'bitter'. The tests were repeated several times.

Taste sensitivity remains practically unchanged until the late fifties when it decreases appreciably in both men and women. Loss of taste is largely the same for all four qualities, although the decline in the 'bitter' sensation seems to be least pronounced. No differences could be found between smokers and non-smokers. The decline in taste sensitivity is presumably due to degenerative changes in the receptors, as anatomical studies performed by other authors have shown.

[Cooper, R. M., Bilash I. and Zubek, J. P. (1959): *The Effect of Age on Taste Sensitivity.* J. Geront., **14,** 56].

Poisoning Caused by Pentachlorophenol

A report is given on 9 deaths which occurred among workers in sawmills in Sarawak (Borneo), where timber is dipped into tanks containing pentachlorophenol as a wood-preservative. The symptoms of poisoning take the form of prolific sweating, hyperpyrexia, dehydration, and dyspnoea. Terminal spasm also develops. The most striking aspect of such cases is the rapidity of the illness; in 6 cases death ensued within 9 hours. No marked pathological findings were evident at the post-mortem examination, except for marked pulmonary oedema. Treatment for pentachlorophenol poisoning was confined to symptomatic therapy, such as measures to combat hyperpyrexia and replace the depleted electrolytes, glucose-infusions, etc.

Following these deaths, strict precautions were taken. Labourers are no longer allowed to spend more than 14 days working at the dip-tanks; rubber gloves, aprons, and boots must be worn; and washing facilities are provided next to the tank so that workers can wash themselves frequently. The strength of the pentachlorophenol solution has been reduced to a

maximum of 2%. In addition, workers are required to drink a pint of milk daily.

The author suggests that the danger of pentachlorophenol poisoning may well be greater in tropical countries, where workers live on a simple rice diet and possibly have some racial susceptibility.

[Menon, J. A. 1958): *Tropical Hazards Associated with the Use of Pentachlorophenol.* Brit. Med. J., **1**, 1156].

Mistakes by Drunk Drivers

The most frequent mistake made by the *motorist under the influence of alcohol* is to 'wobble' from side to side; less frequent are mistakes in gear changing or bad cornering. The errors are particularly numerous on easy stretches of road where the ability to concentrate declines and indifference or lack of inhibition occurs. The number of errors is reduced, albeit for only a short time, on narrow and difficult stretches.

[Abele, G. (1959): Zbl. Verkehrs-Med., **5**, 34].

Colour Films and Occupational Hazards

Laboratory assistants, whose task it is to develop colour films, may suffer from lichenoid skin manifestations or genuine lichen planus as an occupational disease. The noxious agent may be any one of a number of para-phenylenediamine derivatives.

[Graciansky, P. de, St. Boulle, P. Quercy and J. L. Cardot (1959): Presse Méd. **67**, 374].

Reprinted from the Journal of Forensic Medicine, *Vol. 7, No. 1, January-March 1960, pp. 5-92.*

British Association in Forensic Medicine

The 16th Meeting of the British Association in Forensic Medicine was held in Cambridge on Friday and Saturday, 27–28 June 1958.

The Master and Fellows of Downing College kindly allowed members to stay in college for this meeting and most members arrived in time for dinner on the Friday night.

The Council meeting was held in the music room after dinner.

Professor Dean allowed the use of his Department for the scientific meeting on the Saturday.

At dinner that evening the official guests included Professor Guthrie, the Master of Downing, Professor Dean, Professor of Pathology in the University of Cambridge, and Mr. F. Lawton, Q.C., Recorder of Cambridge, as well as many private guests.

Fifty-five members attended the scientific meeting and 57 were present at the dinner.

A symposium on *Identification of Human Remains* was held in the Department of Pathology, Cambridge, on 28 June 1958.

Morning Session: Dr. F. E. Camps (*President*): *Opening Remarks.*
Dr. Deslypere: *Assessment of Age by the Measurement of the Haversian Canals of Human Bones.*
Professor Moureau: *Histological Determination of the Age of the Embryo.*
Dr. R. Fearnhead: *Dental Identification.*
Dr. W. Davidson: *Sexing from Cells.*
Dr. W. E. Evans: *Hairs.*

Afternoon Session: Dr. F. E. Camps: *The Future.*
Dr. F. Fiddes: *Application of the Percentage Cooling Rate Method.*
Dr. A. Warrack: *Asphyxia from Paraffin Heaters.*
Dr. G. Forbes: *Paraffin Granuloma of the Lung.*

Symposium on Identification of Human Remains

Opening Remarks: Introduction

Dr. F. E. Camps

The identification of an alleged human, whether living or dead, fresh or decomposed, part or whole, or even animal, may be one of the most challenging aspects of forensic medicine. It is also, I would suggest, on occasions beyond the scope of any single individual, although this may not have been appreciated in the past. It has been the centre-piece of many famous trials, such as the Tichborne claimant, Adolf Beck, and, to come to more recent days, the Ruxton case, the Setty case and the Haigh case. It has also been the basis of social and administrative problems such as the mixing of babies in a maternity unit nursery and the alleged smuggling of a male child into the parturient bedroom of a noble lady.

It is therefore with no apologies to-day that I open this symposium and, knowing the aspects of the problem to be touched upon by the speakers, I propose to summarize the matters upon which they will not touch.

The identification of the living, moving person on first sight should be simple compared with that of the dead decomposed body. Strangely enough this is not so, for in the former there may be only a fleeting image or impression upon the memory, whilst in the latter there is unlimited time even if less material. Further, the living person may deceive on external appearances whilst the dead cannot conceal the most intimate details of anatomy. Nevertheless basically, identification must be made from investigations.

A Scheme for Identification

A. *Fundamental Facts*:

1. Age: anatomy, teeth, pathological changes.
2. Sex: anatomy (bone, soft tissue).
3. Height: measurement, estimation.
4. Race: anatomy.
5. Finger prints; foot prints; palm prints.
6. Blood groups.
7. Serology (human).

B. *Topographical*:

1. *Face and Head.*

(*a*) *Eyes*: colour, shape of pupil, spectacles (Pathological: cataracts, etc.).
(*b*) *Nose*: shape (Pathological: polypi).
(*c*) *Mouth*: shape, lips, scars.
Teeth: shape, appearance, colour, notching, gaps, no eruptions, fillings, caries, dentures.

Tonsils: present or removed.

(*d*) *Ears*: shape, deformity (cauliflower) (Pathological: discharge from middle ear disease, tophi).

(*e*) *Hair*: colour, texture, quantity, dye, comparison.

(*f*) *Shape of Head*: measurements, anatomy, superimposition photographs.

(*g*) *Special Peculiarities*: scars, moles, blueing, etc.

2. *Trunk.*

(*a*) Shape (configuration).

(*b*) Clothing (size, nature, laundry marks), property.

(*c*) Tattoo marks, scars, naevi, moles, warts, occupational marks, lineae striae.

(*d*) Circumcision.

3. *Limbs.*

(*a*) Size of arms and legs; size of hands and feet.

(*b*) Occupational marks.

(*c*) Deformities: scars, tattoo marks, skin staining (nicotine).

(*d*) Social status and occupation; condition of skin and nails.

(*d*) Property; rings, etc.

C. *Pathological Changes*:

1. *Specific conditions* which may be known, such as gall stones, gastric ulcer, neoplasm, aneurysm, congenital heart disease, old cardiac disease, fibroids, skin disease.

2. *Operative procedures*: scars with absence of organs, stomach, testis, lung, appendix, breast.

3. *Accidents*: deformities, old fractures, old amputations, foreign bodies, scars.

4. *Pathological changes indicating age*: cardiovascular disease, arthritic changes, etc.

5. *Specific pathological changes*: malaria, sickle cell anaemia, special haemoglobin.

From this by no means comprehensive list, it can be seen that special facilities must be available such as radiology, serology, odontology, photography, even chemistry. So too, it is evident that the possibilities of new advances or discoveries must be instantly explored, as is shown by the serological aspects in particular. Dr. Davidson and Dr. Fearnhead will talk about two of these and I do not underestimate Professor Moureau and Dr. Deslypere.

Finally, there is one aspect of identification which is probably not fully appreciated, viz characteristic postures and movements such as gait and habit spasms. Every experienced police officer appreciates the fallibility of the eye-witness, especially over identification, and I had hoped to have Professor Schroeder here to-day to show you his film of a bank robbery which illustrates this only too well, especially the manner in which one of the police officers identified a woman.

I hope that I have covered most of the ground, at any rate sufficiently to give a background for the subsequent discussion.

The Assessment of Age by the Measurement of the Haversian Canals of Human Bones

A Critical Study of the Balthazard and Lebrun Method

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*Laboratory of Legal Medicine of the University of Ghent, Belgium**

The text of this communication was published in this Journal, Vol. 5, No. 4 (October-December), pp. 195–199.—EDITOR.

* *Director*: Prof. F. Thomas.

Identification and Age Determination of Foetal Debris

Prof. P. Moureau and Dr. G. Desoignies
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We present this paper because we think it is infrequent that one has the opportunity to proceed to as many investigations on curettage products as in the present case. Amid blood clots and placental or other debris, we discovered foetal fragments.

First Fragments. Examined under the binocular microscope, the first fragment turned out to comprise part of a lower left limb consisting of part of a femur with leg and foot attached. The toes were completely developed. A higher magnification showed the nails not to be present. A fragment of the pelvis and another of the right femur were also detected. A penis, already well recognizable as such, was present in the perineal region. The tibia had a length of 9–10 mm. The formula of Balthazard-Dervieux applied to the tibia gave a height of 13.8–14.5 cm. for this foetus. This height corresponds to an age of 77.5–81.2 days, or a mean of 79.3 days. When, on the other hand, we used the formula of Troll based on the foot length (here 6 mm.) we obtained an age of 78.4 days.

Second Fragment. This consisted of part of the head, viz. the palate, the upper lip, the nose, the eyes and the anterior half of the skull. They eyelids were closed and the eyes bulging. The nose and the mouth were well separated. This only occurs from the seond month onwards.

Third Fragment. This consisted of placenta, with a stretch of umbilical cord, 2.5 cm. in length, attached. The differentiation of the placenta authorized us to say that this feotus was at least in the third month of its development.

Fourth Fragment. Diverse non-identifiable debris were submitted to a histological examination.

The curettage took place 3 days after the miscarriage; certain of the debris was more or less decomposed and infected.

(*a*) The examination of numerous portions of the placental debris showed that no pathological alteration was present.

(*b*) Part of a kidney with foetal structure was present. A few glomeruli were already formed but decidedly immature; others were limited to a curled-up tube. Only a small number of tubes, spread in a very cellular blasteme were visible.

(*c*) Near the kidney, and in close connexion with it, there was a transverse section of a vertebra, consisting of a piece of cartilage containing embryonic spinal cord with a wide ependymal canal.

(*d*) One of the islets of cartilaginous vertebra was the seat of beginning enchondral ossification.

(*e*) A fragment of suprarenal gland was also present. In this one could clearly recognize the glomerular, fascicular and reticular layers. The cells were not as yet loaded with lipoids. One also noted foci of extra-medullary haematopoiesis.

(*f*) Striated muscle, cartilage and bone were also present. The appearance of points of ossification allowed the conclusion that the age of the foetus was equal to or greater than 2 months.

Conclusion

The macroscopic examination of the head and pelvis, the anthropometric measurement of the tibia and the histologic study of the organs present allowed us to say with certainty that this foetus was 2.5–3 months old.

Dental Aspects of the Identification of Young Persons

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The dentition of human remains sometimes provides infallible evidence of identity. Characteristics of the dentition and of dental treatment are particularly effective in establishing identity when accurately kept dental records are available. In addition to particulars of treatment, dental records may include plaster casts of the jaws, and radiographs. This is especially true when the skeletal material is of a young person who may have been attending a dental practitioner for treatment, e.g. orthodontics.

In the absence of dental records, apart from special features such as peculiarities of development that might be recognized by relations or friends, probably the most useful data that can be expected from an examination of the dentition are those which can be related to age.

An accurate estimation of age can be made by assessing the extent of completion of the roots of teeth, their stage of eruption and the general condition of the jaws of persons below the age of 21 years. Age assessment of adults over 21 years presents much greater difficulty and entails the consideration of physiological age changes in each of the dental tissues, the rate of which is not known, and which are also commonly influenced and obscured by pathological conditions.

Assessment of age can be particularly accurate in young persons during the period in which the teeth are developing, because the process by which the calcified tissues of the teeth are formed is an intermittent one and, in the case of enamel and dentine, rhythmic alternating periods of formation and rest occur at a known rate. The increments of both tissues form a series of fine lines which can be seen by microscopic examination of thin sections. Fluctuation in the finely balanced mechanism of the calcium metabolism of growing individuals, frequently causes the accentuation of one or more of these growth lines. Birth causes a marked transitory imbalance in the calcium metabolism which is recorded permanently by an exceptionally well marked line, the neo-natal or birth line, in the structure of all the teeth that are formed partly pre-natally and partly post-natally.

Calcification of the tips of the crowns of the deciduous incisors has usually commenced by the fifth month. Between this time and the twenty-eighth week, when the foetus becomes legally viable, calcification of the

remaining deciduous tooth germs has begun. Examination of radiographs of fresh specimens in this stage of development can provide useful corroborative evidence of age. Examination of dried specimens, however, is less reliable since the tiny calcified tips of the crowns often become loosened and are easily lost from their bony crypts.

Most textbooks of dental anatomy and histology include charts showing representative stages in the development of the dentition, the one most commonly used being that of Schour and Massler.[1] The data on which their chart was made were obtained from American whites. It has, however, been shown to be reasonably accurate for English children.[2]

It is occasionally necessary to give an opinion about the age of near-term foetuses and newborn infants. In the latter case, histological examination of sections of teeth could reveal whether a neonatal line was present in the enamel and the dentine. If present, measurement from the neonatal line to the forming surface of the dentine could give a reasonably accurate idea of how long the infant had lived.

Recently Stack (personal communication) has established that 'the total weight of the deciduous dentition was found to increase in a simple logarithmic manner during the second half of foetal life. It reaches a weight of two thirds of a gramme at term. Normal growth rates of the various types of teeth were also calculated.'

The original material was obtained from foetuses that were known to have had a traumatic termination, so that in fact the result was most probably a true representation of normal growth rates. The remainder of Stack's specimens were taken from a pool of pathological specimens from which he observed that the 'dental growth below the normal range was observed in 40 cases out of 130, particularly those with a history of placental failure or maternal toxaemia'.

Frequently the time of eruption is used as a means of assessing age and this feature of development is less constant than the rate of formation of the dental tissues.

The time of eruption of individual teeth is known to vary widely, so much so in fact that it is extremely difficult to establish within narrow limits what can be regarded as normal eruption times. Gleiser and Hunt[3] have made a thorough study of the chronology of development of the first permanent molar in American children and Clements, Davies-Thomas and Pickett[4] have provided valuable data on the times of eruption of teeth in English children. Further studies on the chronology of tooth eruption in other racial groups could provide information of very great value not only to the forensic odontologist but to those concerned with children's dentistry.

The teeth have no morphological characteristics that can be related to sex, nor are there any differences in the rates of tooth development and eruption between the sexes. Hunt and Gleiser[5] have, however, been able to sex

correctly 73% of a sample of 2-year-old children and 81% of a sample of 8-year-old children, by comparing the dental age, with the skeletal age. The estimated age was obtained by reference to Schour and Massler[1] Tables and the skeletal age by reference to data compiled by Greulich and Pyle. Hunt and Gleiser's method is based on the fact that skeletal maturation of the female is approximately one year in advance of that of the male. Hence, if the estimated skeletal and dental ages correspond closely, it is probable that the skeletal remains are those of a male, whereas if the skeletal age is in advance of the dental age the remains are most probably female. This method seems to be one which might well profit from further researches.

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Additional Reading

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Sexing from Cells

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Despite the fact that the sex chromosomes were identified in man over 35 years ago, the technical difficulties surrounding their precise recognition in dividing cells are very great, and include the necessity of obtaining absolutely fresh tissues. For example, much of the original work was done on testicular smears made within 20 seconds of death from execution. This has rather limited the direct application of this method to the problems of sex identification in Man and has created the need for a method of sexing the nuclei in the resting phase.

Although overlooked by medical research workers, the entomologists had known for many years that the XX chromosome complex is so much richer in deeply staining hetero-chromatin material than the other chromosome pairs that it is possible to sex insects from the staining properties of their nuclei.

The accidental discovery by Barr and Bertram of a deeply staining intra-nuclear chromatin mass in the neurons of female, but not of male cats, was immediately recognized as a possible method of determining the sex in mammals and the work was rapidly extended to other tissues, including the skin and mucous membranes, and to other animals, including Man. The sex distinguishing nodule is some 1.2 μ in diameter and, in the skin, characteristically lies closely applied to the nuclear membrane. Not only does this mass stain more deeply than does the rest of the nucleus, but it also is Feulgen positive in contrast to the nucleolus. It is believed that the hetero-chromatin material of the single X in the male is not sufficient to allow it to be distinguished from other non-specific thickenings of the intra-nuclear chromatin.

In applying the Barr method of nuclear sexing to problems of human sex identification, it is usual to employ skin biopsy sections or buccal mucosal smears, but there are nodules in the cells of all tissues and they are particularly well defined in cartilage and the suprarenal cortex. The nodules have even been demonstrated in foetal cells from the amniotic fluid, permitting recognition of the sex before birth.

Using the best technical methods available and cells in perfect preservation, for example from tissue culture material, nodules have been found in very nearly 100% of the cells in the female while almost no structures mistakable for nodules occur in the male. Unfortunately, as the preservation of the tissues and the quality of the technical methods deteriorate, recognition of the nodules becomes more and more difficult. In routine skin biopsies some 50–85% of the nuclei in the female have nodules and

under 15% in the male. In skin sections from post-mortem material, Emery found that 5 of 34 specimens from males gave counts which were falsely high, 2 reaching to between 20–24% while in 3 of the 21 specimens from females the count fell below 25% and in one of these almost to 10%. Nevertheless, even in this material 85.5% of the samples could be correctly diagnosed and only in the group between 10–25% was there difficulty.

The best technique has been described by Barr (Moore, Graham and Barr, 1953, Surg. Gynecol. Obstet., **96,** 641) and includes the immediate fixation of thin pieces of tissue in Davidson's fluid* with well-cut paraffin sections and staining by haematoxylin and eosin or, better, by the Feulgen method. With experience it seems that in skin biopsy material recognition can be 100% correct in normal subjects, but I myself have found some difficulty with liver sections where counts are lower than those recorded in the skin and the differences between the male and female is smaller. In short, therefore, the accuracy depends upon the freshness of the material, the tissue examined and the technical methods employed. While it is possible to be quite definite in the diagnosis of tissue from the female, it may be much more difficult to be sure of the negative recognition of the male.

The alternative method of sexing the nuclei, using the neutrophil leucocytes of the blood, was also discovered accidentally while investigating the cause of the diminished nuclear lobing in the hereditary Pelger anomaly of the leucocytes. The feature characteristic of the female is a small drumstick-like projection attached to the nucleus in a proportion of neutrophils, usually in between 1 : 20 and 1 : 50, but occasionally as high as 1 : 5 or as low as 1 : 200. The head of the drumstick measures some 1.5 μ in diameter and is joined to the rest of the nucleus by a filament. It appears that this structure first arises as a deeply staining chromatin mass on the edge of the nucleus, resembling Barr's nodule, but larger on account of the spreading out of the cells while making the blood films. Once this nodule begins to project from the edge of the nucleus as a sessile nodule it seems that it is forced outwards to become a 'promontory' and finally a 'peninsula' or drumstick, a process closely related to the nuclear lobing itself. It is therefore not surprising that the frequency of the drumsticks and the degree of lobing of the nucleus are closely related. In tissue sections, when neutrophils with drumsticks are recognized in the blood vessels, the characteristic nodule is the same order of size as Barr's nodules in tissue cells, i.e. just over 1 μ in diameter.

In normal persons using the best technique, it is possible to get 100% accuracy in diagnosing the sex. The method I have always employed is to allow a small drop of blood to spread between crossed coverslips by capillary

* This contains formalin, 20 parts; 95% alcohol, 30 parts; glacial acetic acid, 10 parts; distilled water, 30 parts.

attraction alone. These are then slid apart, allowed to dry in air and stained mechanically by the Jenner-Giemsa method. The films are examined for both the sessile nodules and drumsticks of the correct size. I have always hunted until I have found 6 of the characteristic drumsticks, as we suggested in the original publication, and usually this occurs in less than 500 cells, but in a few females (perhaps 2%), although there are the characteristic sessile masses, drumsticks are very scanty and over 1,000 cells may have to be examined to find 6. In disorders of the white cells, particularly where there is an extreme shift to the left with curtailed lobing of the cells, the drumsticks may be very infrequent, and sexing by the leucocyte method becomes quite impossible in the uniformly bilobed cells of the rare Pelger anomaly. In the male, no characteristic drumsticks can be found, although other projections are common and in one cell in many thousands there may be a projection which, on superficial examination, might be passed as a drumstick; but the number of these certainly never approaches 6 in 500 cells.

In poor smears the accuracy diminishes. Soon it becomes impossible to be sure that one is dealing with a male, and in really poor smears it may well be impossible even to recognize any drumsticks at all in blood films from a female.

I have recently carried out a number of experiments to see whether it is possible to recover from blood smears and stains such as one may find on glass, metal, plastic and even cloth, leucocytes in a sufficiently good state of preservation to be able to sex them. There is no doubt that it is possible to recover white cells from such material by the techniques I am about to mention, but the methods require further refinement if they are to become a practical proposition, for so far the work involved is colossal and the accuracy has not been as high as I would like to achieve.

If the smear happens to be on a piece of transparent glass it is not too difficult. A piece of glass can be cut out and the white cells stained by techniques similar to those employed for staining thick films for malarial parasites; that is to say, the red cells are lysed before staining the leucocytes with Leishman or Giemsa stain. In such preparations nodules are recognizable, but the white cells have not been spread as far as they are in conventional blood films and measure almost 10 μ in comparison with the usual 13 μ, so that the drumstick can be anticipated to be nearer 1 μ than 1.5 μ in diameter.

The method I have been using to recover leucocytes from blood smears dried for several days or even for several months on opaque materials (glass, plastics or metals) is to soften the dried blood in equal parts of water and serum for 2 or 3 minutes, then remove the excess fluid and make thick smears of the lysed blood on a microscope slide. This is rapidly fixed in 10% formol water and stained with diluted Leishman or Giemsa stain. In these preparations the neutrophils are well preserved and definite sex

nodules can be recognized in most female bloods. In a deliberate blind test on 12 bloods I was able to recognize 4 of the 6 female specimens quite definitely and all 6 male bloods. The other 2 female specimens were in doubt. In both I found fairly definite nodules such as were not present in the males, but I could not find the requisite 6. One was a poor preparation and, on conventional blood films, the other female had very few nodules.

It is also possible to recover white cells from blood stains on cloth by soaking the stain in a similar mixture, squeezing out as much blood as possible on to a slide and then staining as before. The yield of cells, however, is very poor and so far I have not been successful in recognizing female nodules.

Up to the present, examination of dried smears on glass treated as thick films has been most successful, but I think the other methods are well worth further investigation. The technique requires very considerable experience, and is very tedious, but I have hopes that with improvements it may be of considerable practical importance.

So far I have only spoken about tissues and blood smears from normal persons. It is now well established that the presence of the characteristic female nodules, whether in tissue cells or leucocytes, means that the individual is an anatomical female, while their absence indicates an anatomical male. The differentiation extends to the pseudo-hermaphrodites and the presence of nodules indicates the presence of ovaries, while their absence points to testes. However, 2 genetic snags have been brought to light by nuclear sexing. Although fortunately rare, these affect every method of sexing, whether anatomical or nuclear. In the Klinefelter syndrome an anatomical male with testes and, on a rare occasion, even small numbers of spermatozoa, is found to have female nodules in the tissues and blood cells and a recent chromosome study has shown, in one case at least, the XX chromosome pair. In the Turner syndrome an anatomical female, apart from undifferentiated 'streak gonads', has no nodules in the nuclei. Although these 2 conditions are uncommon, it is possible that such errors could occur in about 1 : 5,000 persons, but so far the information is incomplete.

Another very rare possibility of error is in the blood chimaeras. These occur when in twins the male foetus has received a graft of bone marrow cells from his twin sister early in embryonic life. Owing to the failure to develop immunity to cells implanted at such an early stage, they survive into adult life to produce circulating female blood cells. So far only 3 examples of this condition have been recognized.

Apart from these rare difficulties, if the material is good, the method of neutrophil sexing is accurate. In older smears and recovered material, the difficulty is greater but it may be possible to be reasonably certain that the blood has come from a female. The weakness is still that the male can only be recognized by inference.

Hair

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It is a great surprise to me to be asked to speak, as I thought I was here to-day in the capacity of a listener only, so perhaps I may make just one or two random remarks. There is very little I can say about hair as an identifying feature with which you are not already thoroughly familiar, but there are one or two points which may be of interest.

Is hair human? Are hair-bearing remains of human origin? Generally speaking, it is not very difficult to say with certainty when hair is animal and not human. Sometimes, just occasionally, animal hairs may be very difficult to identify as non-human, though if there happens to be a bit of soft tissue from which the hairs have grown, then the precipitin reaction may help considerably. I should be very interested to hear if anyone has tried the precipitin reaction with isolated hairs, and with what result.

One of the simplest tests for human hair is examination of the cuticular scale pattern, which can be done by making a mould of the hair which is half or less of its circumference. Fairly recently Dr. Wildman, of the Wool Research Laboratories at Leeds, has published a very good method for examining the whole of the cuticular surface of a hair. The hair is gently stretched and rolled against an embedding medium (Wildman: *The Microscopy of Animal Textile Fibres.* Leeds: Wool Industries Research Association, 1954). It takes some practice at first, but once it works it gives the pattern completely round the hair.

A method I am trying to develop is direct microscopy of scales by reflected illumination. With this system, the hair can be rotated and studied and photographed without making a mould ('cast'). Human hair usually has the well-known type 7 scale pattern (Smith and Glaister: *Recent Advances in Forensic Medicine.* Churchill, 1939), with a finely serrated scale margin, except in the foetus and neonate, when the scales have smoother edges. Although it is said that hair from the human adult is always of type 7 pattern, in fact human hairs may have a simpler pattern, unlike the typical human pattern, if the hairs happen to have come from a hairy mole or a papilloma of the skin. Some of these hairs are quite large and it is conceivable that such might become detached and thought not to be human, from the scale pattern above. Another exception occurs in hairs from dermoid cysts (teratomata), but it seems rather unlikely that such specimens would have any forensic importance.

A further investigation of value is examination of transverse sections of hair to determine the nature and distribution of any pigment granules and the size of the medulla. A difficulty in this apparently straightforward technique is the cutting of the hair and, for what it is worth, I have found that of a number of embedding media the simplest and best is gum solution from which frozen sections are cut. This avoids the necessity of softening the hair.

Generally speaking, then, hair can be decided to be human or not. If it is human, how much further can we go towards individual identification? I am bound to say that I do not think we can go very far. If one is dealing with a piece of hairy skin, then the anatomical site of the skin may be deduced, as the various hair-bearing regions are well known, but there is immense variation between some individuals in this respect. If the specimen is believed to be from an individual whose hair pattern is known, then the problem is simplified.

Assessment of age from hair is very difficult indeed once the adult state is reached. I do not know any reliable indications of age from hair from an adult.

The Sex of Hair. It is notoriously difficult, given scalp hair, to decide the sex of its former possessor, although the hair pattern on skin, e.g. from the chin, is likely to help matters. I would be very interested to hear, and perhaps Dr. Davidson might be able to say something about this, whether or not the nuclei in the root of a traumatically lost hair can be used for sexing. I have found two difficulties: one is that of getting material fresh enough, and the other the few nuclei that can be seen.

From the point of view of *personal identification,* where it is known that the remains are likely to be those of a specific individual, then the characteristics of the hair are best judged not by microscopy but by ordinary morphology from the point of view of length and apparent coarseness and colour. As far as colour goes there are one or two pitfalls which are well known. The colour of the subject's hair is judged by ordinary observers on the general impression which is given, and often enough the general impression depends on the summation of the various colours which are, in fact, present. If one examines closely any heads of hair one may find quite a surprising range of colour: white, grey, black, brown and even red fibres mixed up in different proportions in different heads to give in each case a different total impression of colour; so that observing one or very few fibres, from the point of view of colour, may be most misleading when it comes to suggesting what an ordinary observer would have thought that person's hair colour to have been.

A couple of points about *colour.* After a period of burial hair colour may change, and in some cases it becomes a dull brownish red (a 'foxy' appearance) and this may occur quite quickly, in perhaps 2 or 3 months,

and after periods of burial of over 100 years, the hair colour may become black. Perhaps Dr. Mant will tell us a little about his observations on hair colour and periods of burial.

The second point is that it may vary during the lifetime of an individual, the childhood hues darkening and then, perhaps, greying with age. Rarely in this country, but not uncommonly in other countries, malnutrition as in ulcerative colitis and kwashiorkor may change hair colour to a rather smoky red, which reverts to the original colour once the nutritional status is restored. This is rather an unusual point, but it may be relevant in certain circumstances.

Alterations in colour from dyeing or bleaching can usually be detected fairly simply and may on occasions be of considerable help. Incidentally, the time at which the dye or bleach was applied can be estimated by determining the length of the root end of the hair which is undyed or unbleached, but there are some problems there. One particularly important one is that a good deal of dyeing and bleaching is done at home with a 'Do-it-Yourself' kit and the process may not be applied very thoroughly, so that the length of hair affected by the dye or bleach is variable and the undyed or unbleached bit of the root may not represent the part unaffected by virtue of hair growth. Wavy hair should always be looked at because it may have been artificially waved. Sometimes there are changes due to artificial waving. Mostly rather irregular transverse fractures occur, but often enough permanent waving does not produce very much microscopic change. The converse is sometimes met with, again not so commonly in this country, but I am told that it is becoming rather more common nowadays, and that is where straight or nearly straight black hair is found on examination to contain breaks or fractures similar to those seen with artificial waving, and that happens when curly hair, the negroid type of hair, has been straightened artificially.

So far as *ethnic groups* go, there is no doubt that the major groups can be determined by hair. From the head hair, a negroid, mongoloid or caucasoid type can be determined, and the degree of hairiness of regions, particularly the axillary region, is sometimes a guide to an ethnic group.

Reverting to the examination of scales, I hope Professor Viterbo may say something in the discussion about his work on the examination of the epicuticle by means of the electron microscope.

Finally I wonder if anyone here has attempted to get tissue *groups* from the hair. I believe in Italy some work has been done. If it can be achieved then it would be one step towards more positive identification.

Discussion

Professor Thomas (Ghent). As shown with thallium poisoning, there is a pigment which leads to large amounts of this pigment accumulating in the roots. One can diagnose thallium poisoning from the hair. If the person recovers and then is submitted to a second poisoning, then one will find large fragments of conglomerated pigment along the shaft, indicating that poisoning has occurred twice. In chronic arsenical poisoning, where we know that the hair contains large amounts of arsenic, one can see it histologically in the reticulum of the hair.

In Ghent some work has been done on the extra chromosome from vaginal smears which seems to be useful.

Dr. Davidson. I have not actually tried sexing material from individual hairs. If, of course, one had a piece of skin it would be quite easy to get a sex, if it were well preserved. But there are many difficulties. I doubt if any individual hair would show recognizable characteristics, but if one could get a little patch of hair follicle it is quite easy.

Dr. Simpson. I think it very important that we should note that it is an estimation of foetal material that we are making and not an estimation of the actual date of delivery. It is an easy mistake to assume that the predicted date of birth is in fact the date of delivery. I learnt my lesson when a body was discovered in a trunk some 15 years ago, and thought that I could reasonably well presume that the child was about 4 months old. But when the mother was traced in the Midlands she stated that the child was, in fact, 6 months. It had been born 2 months prematurely.

In the last series of teeth we did, we estimated the age by using the method of squares and we predicted it within a week in each case. I must point out that in these cases birth had taken place at a known period and that death was due to trauma. If there is a history of toxaemia or loss of blood then our estimations were set back by a fortnight.

Afternoon Session: Short Papers

The Future*

Dr. F. E. Camps

Fellows, Members and Associate Members of the British Association in Forensic Medicine: As one of the original founders of this Association, I have not only had the opportunity of watching it grow, but also (as Secretary for the first few years and a member of the Council) of watching its teething troubles and the impact that it, as an Association, has on the general policy of the teaching and practice of forensic medicine in the British Isles.

During this period, we have had as Presidents men of international reputation and experience coming from, and quite rightly coming from, the only two schools of forensic medicine which have maintained their original status during the period of deterioration in the status of the subject, Sir Sydney Smith and Prof. John Glaister. Further, by the election of Prof. Douglas Kerr as President-Elect, we can look to the maintenance of this tradition so long as it is allowed to last.

When you did me the honour of electing me your President, you did so to cover a period during which anything might happen to this speciality and you gave me a task which, without your whole-hearted support and complete loyalty to the Association, might prove beyond my limited capacity, viz. preserving the speciality as such. For it may well be that if things progress as at the present, by the time I hand over the badge of office to Douglas Kerr it may have ceased to exist. I do not want you to think that I regard myself as being entitled to decide the policy of the Association. That must be done by the Council, but I do feel that as President I am entitled to put forward my views to the Association for their approval or not—that is democracy. For that reason what I am about to say in my first Presidential address is for the benefit of all members of all categories, and I hope that it may be decided that in the future once a year the President shall talk to all the members and enable them to voice their opinions. This does not mean that I am not still completely in support of the policy which was laid down at the original

* Presidential address at the 16th meeting of the British Association in Forensic Medicine, Downing College, Cambridge, 28 June 1958.

founders meeting, viz. that the Fellows shall be elected only from those who teach and practice the subject.

Your learned and efficient Secretary showed some trepidation when I said that this was to be a Presidential address and was fearful that I might upset some of you. I think that is why the nature of this talk has been sprung on you in this way. That I shall do nothing to hurt anyone's feelings deliberately you can rest assured, but at the same time, the time has come to face up to hard facts, and hard facts they are. Let me tell you now that we were all quite adequately warned by Professor Glaister in Glasgow some years ago and yet not one effort was made to take advantage of the warning. I am not going to say that I entirely agreed with his remedies, but the fact remains that the warning was there. That warning was that as things were going the speciality would slowly disappear. And what has happened? The University of London gave up its examination some years ago. It has been followed by other universities. After showing dismay the General Medical Council appear to have slowly yielded to some form of pressure, or is it inanition, and have not supported their, at first, firm attitude. If this policy is weakened, viz. that it is essential that the subject should be taught, then a certain hard core of medical politicians in universities will take full advantage and do the rest.

Now what is strange about all this is that whilst the situation is deteriorating here, in the United States never have the public, the medical and the legal professions been more acutely conscious of the necessity of improving it. If I may quote a few examples, having seen from bitter experience the danger of pathologists doing medico-legal work without training. The American College of Pathology and the American Association of Clinical Pathologists have insisted that anybody taking up the subject as a speciality must have special training and be boarded before allowing to practise, and that *after* having training in pathology. Further, they have appreciated the importance of proper laboratory facilities. Admittedly they attribute some of the troubles to an obsolete Coroners system and are fighting in many States to get the Medical Examiners system introduced. Let me say here and now, I do not like that system mainly because I do not think doctors make good legal opinions although they may think they do. Let me, however, give them due credit because they contributed to a forensic pathology meeting several hundred dollars as sponsors whilst *no* Association in this country, although willing to sponsor the meeting, paid anything as Associations. It was left to individuals to contribute the money and in some cases this was done in the name of Associations. So, too, on the Continent there is also a revival. It is, in fact, shown by the presence of medico-legists from Belgium here to-day. Thus this general deterioration is taking place when never have conditions or facilities been better.

It is no good producing statements of this kind without evidence, so let me quote you some cases:

1. Recently some bodies were exhumed and the examinations carried out by a pathologist. He described:

Congestion (later called ulceration) of the oesophageal mucosa in a body buried for 12 months. The condition of the mucosa of the small intestine was assessed by looking at it without opening. He said that histology would be of no value in death from coronary artery insufficiency, you could tell that from naked eye—and in a body buried for 12 months. This was only a small part of his evidence which, judging from the expression on the face of one of your more eminent members, was not quite acceptable. But accepted it was. So was the evidence of a chemist who said that 2 mg. of a certain poison was the most he had ever met! When, in fact, in a case not far from him 9 mg. had been recovered.

2. A post-mortem examination was carried out by a pathologist for the Coroner and the cause of death given as coronary artery disease. Later it became apparent that the woman might have been murdered by strangulation. When asked about his examination he quite frankly admitted he had not examined the neck. He had been told by the police there was no suspicion! Why do a post-mortem examination? At the examination not only had the larynx not been examined *but* there were 2 cuts across it carved by the post-mortem attendant who had eviscerated the body. It was quite insignificant that the brain had been called normal without opening the skull and a large fibroid uterus had not been noted in the report.

These are only two; there are many more. How can this occur? Professor Mackintosh at Oxford some years ago said that pathologists were the worst enemies of anaesthetists because they found an excuse for deaths which were rarely due to bad anaesthetics. What do you think would happen to a surgeon if he did such things? I am telling you this because I know that none of you would do anything like it and it is up to you not only to set a standard but also to see it is maintained and to report it if it is not.

Now let us pause for a moment to consider why all these things are happening. My own view is based upon observations not only here but in the U.S.A. and elsewhere, and it appears that this work (upon the accuracy and integrity of which the lives and liberty of individuals depend) requires no qualifications or status. Let me give you a few examples.

1. A *toxicologist* (and I am not sure whether such people exist here; they do in U.S.A.) is a chemist who does analyses for poisons. He is nearly always a very good chemist but he has no knowledge of medicine—that does deter him sometimes, probably, but not always. In the forensic science laboratories his experience is based upon what he has read and

been told. Is he taught? Has he attended lectures? No, there are none. Nor are there many chemists outside the laboratories who can do the work. If they do, they are confronted with a name: a big name, a big organization. It is quite true what one of my friends has written, he has never heard their figures challenged. They are probably not challengeable *but* there is no one to do it if they were. So I think we can safely say that there is no qualification for a forensic chemist.

What is a forensic scientist? He is a scientist who has got mixed up in the investigation of crime. How has he been taught? He has worked in a forensic science laboratory. What has he got that any good specialist in his own line has not got (i.e. a good botanist on grass or seed), but he is an expert. What does he know about stains that a good serologist does not know? But he is an expert: no training, no qualifications.

What is a forensic pathologist? In the past there were a certain number who were self-taught by experience much the same as those in other specialities, but they were not pathologists, only police surgeons. Now if pathologists do the work one had thought that they would want to gain experience from those who had it—many did but one at least said he needed no such advantage. Do I hear an echo? Of course it is the forensic scientist who rang up an expert in a special subject and when he had got the necessary information took it triumphantly to his chief to be told 'we don't require advice, we know!'

I do not need to mention the psychological aspects of the choice of personnel. I have already done this in Brussels last year, but let me repeat that anybody who shows himself capable of self-deception in the small things must be regarded as a risk because he will deceive himself when he makes a mistake. Such self-deception is dangerous because it is not appreciated. I remember very vividly a man coming to me after giving evidence in a big case and telling he how well he had done. Later both prosecuting and defending counsel told me that he did very well up to a point and then made a serious mistake which won the case for one side and lost it for the other, and yet he did not realize it himself.

There is one other aspect upon which I must touch. I call it the 'enemy in our midst.' Again I am sure they do not realize what they are doing or if they were accused of being dishonest would feel dreadfully hurt. All the more dangerous!

Let me give you a few examples—

1. A pathologist refuses to do a post-mortem examination in a certain mortuary because it is unsuitable. To his amazement he finds that somebody else has done it. To cap it all he is asked to give an opinion on the tissues (under his N.H.S. contract). This is similar to Ryden in Galveston, Texas, who was asked to do an autopsy on a (very) decomposed drowning case. Afterwards he told the Coroner he did not know the

cause of death. The Coroner replied he would get somebody who would. He did. What these good-natured, weak, perhaps greedy people do not realize is that they reduce the standard and they prevent it from being raised. In other cases they afford a threat which enables it to be kept low.

But all these things that I have told you have a worse effect. They discourage people from learning the job and training because what is the point of doing it if there is nowhere to use it? Further, they play into the hands of our critics who merely say that it confirms what they thought —a low standard, no interest in other than money or publicity. They forget that when making these epoch-making observations they are effectively arranging a noose round their necks, even if innocent. Perhaps we shall have an eminent person charged with some offence on such evidence.

The Future

Where have we failed?

Personal publicity.

Support of bad practice (i.e.) loyalty to the profession rather than the subject.

Acceptance of employee-relationship with the Coroner.

Acceptance of poor structures of mortuaries.

Failure to enforce discipline and demand qualifications.

Failure to press for qualifications in order to obtain posts, etc.

Fear of unpopularity.

Failure to promote the Association's influence.

Failure to press for teaching posts in Universities and Departments of Pathology.

Failure to demonstrate a high standard of work and clinical improvement.

What we must do.

1. Improve the status of the Association.
2. Request representation in decisions affecting legal medicine, e.g. *cremation.*
3. Press for security for the pathologist in relation to his work with Coroners.
4. Post-graduate classes and certificates of qualifications.
5. Train young pathologists.
6. Broaden the subject (i.e. teachers *must* practise forensic medicine).
7. Co-ordinate ancillary subjects.

* * *

Dr. Fiddes spoke on *Application of the Percentage Cooling Rate Method.* This has previously been published in this *Journal,* 1958, **5,** 2.

Asphyxia from Paraffin Heaters

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Carbon monoxide is a well-known source of poisoning. Simpson[1] states that 98% of all suicidal gas poisonings are due to domestic coal gas, whilst 50% of all accidental deaths from gases arise from the same source. In industry more than half the accidental cases of gas poisoning are also due to carbon monoxide.

Although the commonest source of carbon monoxide is domestic coal gas, many other sources responsible for fatal accidents have been described. These include car exhaust fumes, blast furnaces, coke braziers and even the exhaust fumes from machine guns in pill boxes.

The object of this paper is to describe another not uncommon domestic source of carbon monoxide poisoning.

On 20 November 1954, at 9.35 a.m. the Police were called to a house where a young man had been found dead. They found a young adult male in a natural position in his bed. He was wearing a shirt, vest and socks. There was no evidence of violence.

The room measured 9′ 6″ x 8′ 6″ x 8′ high, i.e. 646 cubic feet. There was one small window which was closed. There was a small air grate high up in the wall near the window. This had been boarded up, apparently for some considerable time. There was no fireplace.

The sister of the deceased stated that he had returned from work at 6.30 p.m. on the previous evening. He had complained of feeling cold and had thought he was going to have influenza. However, he stayed up and watched television, finally going to bed at midnight. On going to call her brother at 9 a.m. the following day she smelt paraffin fumes. On opening the door she saw a paraffin pressure type heater standing on a chest of drawers at the foot of the bed. She stated that it was warm and that it was hissing and giving off fumes. She was sure that it was not burning. There was some paraffin round the base of the lamp. She turned off the lamp and took it out of the room. Her brother was apparently dead.

A post-mortem examination was performed at 11 a.m. on 22 November. The deceased was a well-nourished young male about 26 years old. There was no cyanosis, but the lips were pink. Post-mortem hypostasis was present and appeared pink. There was some fine froth at the nostrils.

On internal examination there was congestion of the respiratory tract and the lungs were oedematous with congested bases.

The heart was of normal size with no valvular disease. The coronary arteries were patent. There were no petechial haemorrhages present in either the respiratory or the cardiovascular system. All the other organs were normal.

Histological examination of the heart and lungs showed no evidence of acute infection and analysis of the blood and stomach contents for barbiturates and aspirin was negative. The blood showed a 20% saturation with carbon monoxide.

At the subsequent inquest the sister corroborated her statement to the Police and identified as her brother's property a paraffin pressure heater. A representative of the manufacturers, in describing the heater, stated that oil was forced by pressure from a small pump into a vaporising tube. Heat from methylated spirits converted the paraffin into vapour which passed through a jet into an asbestos burner. Once the lamp was functioning the heat generated kept the vaporiser hot. The lamp would burn as long as there was a positive pressure in the container. If the lamp went out owing to inefficient combustion, then liquid fuel would hiss out on to the table. This is, in fact, what was described by the deceased's sister. The witness further stated that the lamp at full pressure would use up the oxygen in 36 cubic feet of air in one hour, thus exhausting a room of the dimensions mentioned in 18 hours.

He further said, however, that tests had shown that it would not in fact exhaust all the oxygen but would be extinguished due to shortage of that gas in a very much shorter time. He pointed out that on this particular lamp the asbestos mantle was missing and had been replaced, presumably by the deceased, with a strip of perforated metal. In his opinion this might slow down the process of combustion, producing carbon monoxide instead of carbon dioxide. Correctly used in a ventilated room the lamp would not produce carbon monoxide.

It being noted that the carbon monoxide saturation of the blood was only 20%, the final verdict was 'Death from asphyxia due to lack of oxygen and the presence of carbon monoxide in the atmosphere, and further that asphyxiation was due to misadventure through sleeping in an unventilated confined space with a paraffin radiator burning, which was made defective due to an alteration in the construction made by the deceased'.

This case therefore leaves us in some doubt both as to the part that carbon monoxide played in this death and as to the question of how dangerous a lamp of this type can be. This one had been modified and, as I have indicated, the possible significance of this was not missed by the manufacturers' representative.

The next case seems to leave less room for argument.

On 24 December 1956, at 6.35 a.m. the Police were called to a lodging house where a married couple had been found dead. They found two Jamaicans occupying a double bed. The man was lying in a natural position with his head on one side. He was wearing a shirt, a vest and 2 pairs of pyjama trousers. The woman was curled up on her right side across her husband's feet as if she had been trying to get out of bed. She was wearing a head scarf, a nightdress, 2 skirts, a pair of knickers and some thick woollen socks.

The room measured 12′ x 8′ 6″ x 8′ high. The window was of the sash type and tightly shut. The door was described as fitting with no gap at the bottom. There was an air grate near the ceiling. It had been closely sealed by a metal plate. There was no fireplace.

The lodging house keeper, also a Jamaican, said that the deceased couple had gone to their room at 8 p.m. on the previous night in normal spirits. At 6.25 a.m. his wife had gone to call them and had smelt paraffin fumes. The witness had been summoned and had forced the door. It was the usual practice of the deceased couple to lock their door. On entering the room he saw a paraffin pressure stove on the floor at the foot of the bed. It was hissing and warm and was giving off dense fumes. He thought that it was also burning with a pale blue flame. He did not remember if there was any spilt paraffin in the vicinity.

The Police reported no signs of violence and a tin trunk under the bed containing £120 in cash and Post Office Savings Credits for about £200 was intact.

A post-mortem examination was carried out 24 hours after death. The male was well-nourished and about 40 years of age. The colour of the post-mortem hypostasis was difficult to elicit but the conjunctivae were pink. Internal examination showed unusual pinkness of all the organs. The trachea and bronchi contained pink, frothy mucus. The lungs were congested. The heart was normal and no petechial haemorrhages were found. There was no abnormality in other organs.

The findings in the woman who was aged 34 were essentially similar.

Histological examination showed no evidence of any infection of the heart or lungs in either case and analysis of blood and stomach contents was negative for alcohol, barbiturates and aspirin. The carbon monoxide saturation of the blood was 70% in each case.

At the inquest the story of the lodging house keeper was confirmed and he identified as the deceased couple's property a bowl-type pressure paraffin heater. This was of different manufacture but worked on exactly the same principle as that described in the first case.

A representative of the manufacturers again explained the working of the lamp. He stated that this one was normal in all respects. He was certain on the evidence he had heard that the lamp had gone out just before the

room was entered and that it was likely that insufficient air had been available to make combustion complete. Carbon monoxide would be produced as a result. He said that this type of lamp used the oxygen in 40 cubic feet of air in 1 hour.

The verdict in both cases was 'Asphyxia due to carbon monoxide poisoning and further that the deceased died by misadventure through sleeping in an unventilated room heated by a paraffin heater which was in good condition but produced carbon monoxide from incomplete combustion by reason of the exhaustion of oxygen from the air'.

This case therefore seems to confirm the more than strong suspicion that in the first case the modification to the heater was not in fact responsible for the production of carbon monoxide but that these heaters, when burnt under conditions of poor ventilation, are lethal.

In order to try to confirm this a heater of the type concerned in the first incident was procured but in this case the asbestos burner was as supplied by the manufacturers. It was lit and placed in a sealed fume cover of 50 cubic feet capacity.

The lamp burnt efficiently from 9.15 a.m. to 10.30 a.m., but after that the flame became less intense and the mantle ceased to glow. Burning continued, however, until 8.50 p.m. when the lamp went out. At this time blue flames were given off and paraffin poured from the jet.

During this period samples of the air in the chamber were taken. These were analysed in an infra-red carbon monoxide analyser by my colleague Dr. Veral of the University of Sheffield, Department of Medicine, with the results shown in Table 1.

TABLE I

Time	*Carbon Monoxide*	*Carbon Dioxide*
12.45 p.m.	0.0097%	0.48%
8.30 p.m.	0.047%	0.58%
9.10 p.m. (The lamp had been extinguished for 20 minutes)	0.072%	2.4%

The figures for carbon monoxide were, in my view, disappointingly low considering that the capacity of the fume cover was only 50 cubic feet. It may well have been, however, that if the sealing of the door had been less efficient perhaps more carbon monoxide would have been produced. In any event there seems no doubt that under conditions of poor ventilation a lethal concentration of the gas can be produced.

The two incidents, therefore, had certain points in common. A pressure type paraffin heater was used. The rooms were poorly ventilated when the deceased were discovered. The lamps had only just, or were on the point of going out and blue fumes were coming from them.

Both manufacturers' statements and my own experiment indicate that these fumes had nothing to do with death in that they only occurred at the end and were an indication that there was still some pressure left in the reservoir.

At both inquests H.M. Coroner suggested that it would be wise for manufacturers of these lamps to issue a warning in the package against their use in unventilated rooms. I am not aware that this has been done.

I would like to thank H.M. Coroner for the City of Sheffield for permission to report these cases and Dr. Veral of the University of Sheffield who carried out the carbon monoxide air analysis.

Reference

1. Simpson, K. (1958): *Forensic Medicine*, p. 314. London: Edward Arnold & Co.

Paraffin Granuloma of the Lung

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Liquid paraffin has, for various reasons, been under suspicion at different times, since it began to be used to any extent medicinally at the beginning of this century. It has been charged with preventing intestinal absorption of food in general and of vitamin A and its precursor, carotene, in particular. On the first count, there is a conflict of evidence; on the second count, the evidence shows that its administration close to meal-times, or its use in the preparation of food, does interfere with intestinal absorption of vitamin A or carotene, but there is a critical level (2.5 c.c. *t.i.d.*) below which this interference is insignificant. Its use independent of meals, for example the usual therapeutic dose of 30 c.c. given at bedtime, was found to be without any effect in this respect.[10]

Since liquid paraffin is a mixture of liquid hydrocarbons obtained from petroleum, the possibility that it might contain carcinogenic fractions has been investigated. Attempts to produce gastro-intestinal cancer in animals by its use have always failed.[2] However, the same authors (comparing the purgative histories of a series of patients with gastro-intestinal cancer and those with non-gastro-intestinal disease) found that there was a marked excess in the purgative use of liquid paraffin amongst patients with gastro-intestinal cancer, and concluded that liquid paraffin might play some part in a small proportion of cases of gastro-intestinal cancer in man.

At one time there was a practice of instilling liquid paraffin into the peritoneal cavity, with the avowed object of preventing adhesions. The result of this practice has been to produce, in a number of cases, an oil granuloma of the peritoneum.[1]

Probably, however, the most common danger arising from the medicinal use of liquid paraffin, is the production of oil aspiration pneumonia.

The pneumonia which results from aspiration of oils and fats is a subject which has been dealt with extensively in the American literature. It has not received the same attention in Britain, although it is thought that the incidence of the condition in this country may not be any less. The diagnosis of the condition in life is not easy, since similar lesions may be produced by other agencies, and it is felt that fats and oils are not often considered as aetiological agents, because of a fairly general lack of aware-

ness of their ability to produce such lesions, and a failure to elicit, in consequence, the relevant facts in the patient's history. In the cases described in this paper, aspiration of oil was not considered in diagnosis during life. The diagnosis at autopsy also presents some difficulty. The lesions produced may easily be confused with those of a number of other conditions. The essence of the diagnosis here, also, is an awareness that oils and fats can produce a pneumonia and, in suspected cases, to stain sections of lung tissue for fats.

Ikeda[5] classified oil aspiration pneumonias into two types, which he called 'adult' and 'infantile'. The distinction does not appear fundamental or clear-cut. The 'infantile' type occurs in debilitated children and adults as a result of such procedures as forced or artificial administration of food and drugs, containing such oils and fats as cod liver oil, milk, cream, liquid paraffin and others in common use. This type is described as essentially a diffuse interstitial pneumonia of low grade, punctuated with exudative phases, which may be due to the irritative character of the oil or to secondary infection. One of the main grounds of distinction from the 'adult' type is that it may undergo resolution. The 'adult' type occurs among the older age groups as a result of habitual self-administration of liquid paraffin, either orally or intranasally, in large total amounts. Animal oil, significant in the cause of the 'infantile' type, is of little importance in the 'adult' type. Vegetable oils are very rarely implicated. The lesion is described as a chronic granuloma, characterized by areas of dense fibrosis of the lung.

The character of the aspirated oil, its total amount, and the duration of exposure, seem to be fundamental factors in the production of a reaction in the lungs, and in the nature of that reaction. Pinkerton,[7] in a study of the effect of intra-tracheal injection of various kinds of oils in puppies and rabbits found:

(1) That with the exception of chaulmoogra oil, which caused acute necrosis of lung tissue, most vegetable oils remained in the lungs for months without producing any reaction; and

(2) That animal oils and mineral oils produced a similar type of reaction. In the early stages there was a patchy consolidation, found microscopically to be produced by the stuffing of the alveoli with large mononuclear cells, which had taken up the oil. These mononuclear cells thereafter fused to form giant cells, and simultaneously there was connective tissue proliferation. The final stage was one of dense fibrosis, which enclosed in its meshes free pools of oil, due to the coalescence of droplets released by disintegrating mononuclear cells.

The course of the reaction appears to vary as between the different oils. Generally the stages of the reaction produced by animal oil appear to be more telescoped in time. The intensity of the reaction as between animal

oils appears to depend on the amount of free fatty acid originally present. Animal oils appear to undergo changes suggesting disintegration, whereas mineral oils remain unchanged in the tissues. Fibrosis appears to be more marked with mineral oil. Since Pinkerton's study, it must be added that one vegetable oil, viz. iodized poppy seed oil (used for bronchograms) has been incriminated on a few occasions, as the cause of oil granuloma.[3]

Among the factors predisposing to aspiration of oil into the lungs are the feebleness asociated with early infancy and old age, the disabilities arising from affections of the central nervous system, debility, recumbency, dysphagia, and lesions of the mouth, throat, oesophagus and upper respiratory tract. The character of the oil also is an important factor. Liquid paraffin is implicated as the causal agent in the great majority of cases. Its blandness would seem to be the reason for its known capacity to enter the bronchial tree without exciting any reflex action. The type of case in which oil aspiration pneumonia is most likely to be found is the chronic sufferer from respiratory infection who, for long periods, uses oral or nasal sprays, of which liquid paraffin is an ingredient; or the sufferer from chronic constipation, who seeks relief in a regular oral dose of liquid paraffin over long periods.[4]

Pathological Changes

The condition is comparable to others whose main effect is to produce fibrosis of the lung tissue, e.g. pneumoconiosis. All grades of severity may be found at autopsy. There may be an incidental finding of a few discrete nodules, causing little or no symptoms in life. On the other hand there may be extensive involvement, causing serious impairment of pulmonary ventilation, and pulmonary vascular hypertension. There cannot be said to be a typical naked-eye appearance. There are irregular areas of consolidation, varying in size from 1 cm. or less in diameter to that of a whole lung segment. The lesions, being fibrous, have a tough or hard consistency. They are usually grey or yellow in colour, and dry. On naked-eye examination and radiologically,[9] they have been most often wrongly diagnosed as neoplastic infiltration or as the caseous consolidation of tuberculosis. They have sometimes been mistaken for patches of unresolved pneumonia. The true diagnosis is very often not even suspected from the gross appearance, and it must be admitted that, in many cases, it is only as a result of the somewhat puzzling appearance of the microscopic sections that suspicion falls on the true agent.

The microscopic appearance depends upon a number of factors, of which the type of oil, the length of time over which it has been administered and the total amount aspirated, appear to be the most important. Different fields may show different stages of reaction. In some areas there may be

found an accumulation of phagocytes in large numbers, occupying the alveoli. The appearance of these arouses suspicion. They are large, foamy, vacuolated in varying degree, even shaped like a signet ring, depending on the degree of subdivision of the fat which they contain, and the amount of decentral displacement of the nucleus. Strands of collagen fibres may be seen insinuating between these lipophages. In other areas are to be seen necrotic foci, with giant cells, formed by the coalescence of the lipophages, and in these areas the appearance may be one of extreme disorganization. In yet other areas there is dense fibrosis, with lacunae of circular shape and varying size lying in the fibrotic mass. Appropriate stains show these lacunae to be, in fact, the oil pools already referred to and which, with routine staining methods, appear as lacunae. Their presence raises suspicion about the real nature of the lesion.

As might be expected, there is a number of complications, which will each add its quota to the appearances described. Bronchial obstruction, resulting in obstructive pneumonitis, recurrent broncho-pneumonia, bronchiectasis, abscess formation and empyema have been known to complicate the disease. Bronchogenic carcinoma has been suggested as a complication, but no satisfactory evidence has yet been adduced to demonstrate any causal connexion. Experimental work has suggested that the lesions might predispose to tuberculosis, but this has not been shown clinically.[6]

The crux of the diagnosis lies in the demonstration of lipoid material in lacunae in the densely fibroid lung tissue. These are demonstrated in the first instance by staining frozen sections with such stains as Sudan IV and Nile blue sulphate. To distinguish between mineral oils on the one hand, and vegetable and animal oils on the other, use has been made of the reaction of osmic acid, which is reduced by the animal and vegetable oils to give a black precipitate, whilst mineral oils have no such action. Some investigators have carried out chemical analysis of lung tissue.[6, 11]

Case Histories

Case No. 1. Mrs. G. F., aged 54, was admitted to hospital on 17 January 1956, with a diagnosis of bronchitis, bronchiectasis and right-sided heart failure. There was a history of chronic cough since childhood. An X-ray report at that time mentioned gross bilateral emphysema, secondary to chronic bronchitis, with bilateral bronchiectasis in the lower lobes. Investigation for tuberculosis was negative.

On admission she had marked dyspnoea at rest. There was diffuse broncho-spasm and expiratory and inspiratory rhonchi, with coarse crepitations at both lung bases. The jugular venous pressure was raised. There was oedema of the ankles and sacrum. Laboratory investigation of the sputum showed normal flora, and 2 specimens were negative for acid-fast

bacilli. The urine was sterile on culture, and apart from a trace of albumin was not otherwise abnormal. The blood urea was 47 mg. per 100 ml. Haemoglobin: 87% (Haldane). White cell count total: 10,400 per cu. mm., with a normal differential count. Chest X-ray report on 26 January 1956 was a repeat of the previous findings, but in addition there was general enlargement of the heart, and the appearances suggested myocardial failure.

The patient responded temporarily to treatment, but died on 7 February 1956.

At autopsy, the main findings were as follows:

The abdomen was distended, and the ankles and legs oedematous. The trachea and bronchi were filled with frothy mucus. Both lungs showed diffuse emphysema and extensive areas of patchy consolidation, particularly in the lower lobes. There was marked prominence of the bronchi and bronchioles. The right ventricle was hypertrophied. The liver on its cut surface showed a nutmeg pattern. The peritoneal cavity contained 2 pints of serous fluid.

Histological examination of the lungs showed the changes of chronic bronchitis and emphysema. In many areas the alveoli were filled with large, foamy, pink macrophages, many of which contained 2 or more nuclei. In other areas, there was a picture of extreme disorganization, characterized by the presence of giant cells of bizarre shape, suggestive of foreign material in the lung tissue. In yet other areas were large collections of lymphocytes and plasma cells. There were also extensive areas of fibrosis, in the meshes of which, on staining with Sudan IV, were revealed a number of oil globules of varying size. These oil globules were found not to reduce osmium tetroxide. It was therefore concluded that the findings disclosed the presence of an oil aspiration pneumonia, in both acute and chronic stages, due to liquid paraffin.

Enquiry subsequent to the post-mortem examination yielded no information about the source of the aspirated oil. It was surmised that it was probably an oral or nasal spray, containing liquid paraffin.

Case No. 2. This and the following cases were subject of autopsies undertaken on behalf of H.M. Coroners for the City of Sheffield and The County Borough of Rotherham.

G. W., aged 55 years, a universal grinder in a steel foundry, died suddenly on 1 November 1957. For several years he had been under the care of his doctor, on and off, because of pains in his chest, shortness of breath and chronic cough. He had, in fact, been treated for bronchitis. Three weeks before his death he was seen by his doctor because of an attack of 'influenza' and had never fully recovered.

The main findings at autopsy related to the chest. There was a quantity of muco-pus in the trachea. In the lower lobe of the left lung were 2

dense fibrous masses, each about 1 inch across. There was a similar mass just above the hilum of the right lung. There was no significant emphysema. The heart was substantially dilated and hypertrophied on the right side. Pulmonary fibrosis was present and an important point was whether it was of industrial or non-industrial origin.

Histological examination revealed varying degrees of fibrosis throughout the lungs. In many areas the appearance was of a honeycomb of clear spaces which, by staining with Sudan IV, were shown to be filled with fat. Osmium tetroxide was not reduced by this fat.

The framework of the honeycomb was of fibrous tissue, but this varied in appearance from area to area. In some areas this framework was of wide, dense, relatively acellular fibrous tissue, and here the clear spaces were largest in diameter. In other areas the fat-filled spaces were smaller and the fibrous tissue framework was more slender and relatively cellular, the cells being a mixture of fibroblasts and vacuolated macrophages, also shown to be filled with fat. Occasional giant cells were to be seen in this fibrous framework. In some areas the normal alveolar pattern was to be seen, many of the alveoli containing numbers of the large macrophages in various states of vacuolation, which we have learned to associate with lipoid pneumonia. Here and there throughout the lung fields were collections of lymphocytes and plasma cells. The basement membrane of the bronchi was thickened. There was no evidence of silicosis or tuberculosis. There was dramatic narrowing of the branches of the pulmonary artery, with marked medial thickening. This was seen to a lesser extent in those areas of lung tissue only slightly affected by fibrosis.

The cause of death was cor pulmonale, due to pulmonary fibrosis, due to lipoid pneumonia. In view of the findings an effort was made to ascertain the source of the aspirated oil. It was found that he had been in the habit of taking liquid paraffin as a purgative. This was the only ascertained source from which the lesion could have arisen.

Case No. 3. J. H., aged 78 years, had worked in various coal mines all his life. Since the age of 70 he had had at least 2 strokes, from which he had made substantial recovery. For a number of years he had had shortness of breath, cough and chest pains. By July 1957, when he was admitted to hospital for a minor condition, he could do little more than totter about owing to senility. He was kept in hospital because of the difficulty of home nursing and, while there, a chest X-ray was reported as showing nodular opacities in both lungs due to pneumoconiosis. On 11 December 1957, while sitting in the ward, he had a sudden pain in the left leg, and on examination presented all the signs of arterial blockage in this limb. Gangrene of the limb developed. He died on 13 December 1957.

An autopsy was undertaken to determine whether pneumoconiosis was the cause, or a contributory factor in the cause of death. He was found to have died of broncho-pneumonia, due to gangrene of the left leg, due to femoral artery occlusion and arteriosclerosis. An incidental finding was that in the lower part of the middle lobe of the right lung there was a fibrous honeycombed area, which was not pigmented, and the appearance of which suggested that it might be an oil granuloma.

Histological examination of this area showed that 3 stages of reaction could be distinguished. In some fields the alveolar septa could be made out and within the alveoli were collections of cells, showing various degrees of vacuolation, most of them being, in fact, of signet ring shape. Sudan IV staining showed that they were filled with fat. Their nuclei and cell membranes were in various stages of disintegration. Fine fibrous strands were just beginning to make their appearance, insinuating amongst these lipophages. In other fields the alveolar pattern was obliterated, and there were sheets of these signet ring cells, distended in varying degree with fat, interspersed here and there with not very large clear spaces, which also proved to be filled with fat. In this type of field fibrous bands were more obvious, and giant cells were also present. In yet other fields, the picture was of a honeycomb of large clear spaces of varying size, shown to be filled with fat. Osmium tetroxide was not reduced by this fat. The framework of the honeycomb was dense fibrous tissue.

As far as the relatives knew, he had not been in the habit of taking liquid paraffin. He was described, however, as being always constipated, and taking things for that. It may, therefore, very well be that he was in the habit of taking this substance unknown to his relatives. It is surprising how little is known sometimes by the relatives of the habits of older people.

Case No. 4. F. F., a widower aged 79, a retired pattern maker, died suddenly on 14 February 1958. He had been in failing health for some time, and had been under the care of his doctor in the recent past, having had two attacks diagnosed as cerebral thrombosis; but at the time of his death he had not been seen by his doctor for some weeks.

At autopsy, he was found to have generalized arteriosclerosis, with 2 areas of old softening in the left cerebral hemisphere. The lungs showed broncho-pneumonic change and, indeed, the cause of death was given as broncho-pneumonia, as a consequence of arteriosclerosis and cerebral thrombosis. In the lower lobe of the right lung a patch of consolidation was discovered, which was larger and had a much harder consistency than the surrounding patches of broncho-pneumonic consolidation.

Histological examination of this area revealed a picture similar in essentials to that described in Case 3. Sudan IV staining showed the area to contain a large amount of fat, which failed to reduce osmium tetroxide.

Discussion

For practical purposes, the agent responsible for oil aspiration pneumonia in adults is liquid paraffin. It gains access to the lungs most easily by its use as an ingredient in nasal or oral sprays. Some years ago it was commonly found in such sprays, and we cannot but feel that there must be in existence quite a crop of oil granulomas of the lung arising from their use. The evidence suggests that, once in the lung, liquid paraffin remains as an inert foreign body for many years, and probably permanently. From enquiries made it appears that the tendency is for nose drops and nasal or oral sprays to be made up in an aqueous medium. A substantial proportion, however, is still being manufactured in an oily medium, containing liquid paraffin. It is estimated that about 20 per cent of nose drops and sprays sold contain liquid paraffin. Their manufacture in an oily medium should be stopped. It is also clear that liquid paraffin gains access to the lungs as a result of oral administration. Naturally, the longer the period over which it is administered, the greater the chance and the quantity. It has been often repeated that those at risk are the old, the young, and those suffering from some disability which allows the oil to slip down into the larynx and the trachea. There are suspicions that the risk is not entirely eliminated even in those who suffer no such disability. A bland oil introduced by catheter into the pharynx of a healthy adult whilst asleep on his back, was shown to be present in the lung on the following morning.[8] My information is that there is still a steady sale for liquid paraffin as a laxative in this country, and I feel that its use for this purpose requires reappraisal.

It may be said that oil aspiration pneumonia is so uncommon as to make such a reappraisal unnecessary. The number of deaths in which it is a contributory cause is very small. No doubt (it may be said) there is a number of people with an oil granuloma of the lung, which is found incidentally at autopsy, and which has made no significant contribution to the death. I can only reply that oil granuloma of the lung is preventable; its presence is known to dispose to recurrent respiratory infection; and in these days when our lungs have to contend with so many other noxious substances, this is one lesion which, however small, we can well afford to do without. Moreover, it may be commoner than is generally supposed. It is probably overlooked frequently.

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Morning Session

Contact Traces

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In my car I keep a post-mortem bag, a scene-of-crime bag, a case of empty, chemically clean bottles and protective clothing—both for the post-mortem examination and the weather. This is because I have always been taught, and I endorse this teaching, that the investigation of a scene is ideally made by the investigating authority (viz. the Police), the photographers, the fingerprint experts, the Forensic Science Laboratory (represented by the Police, Liaison Officer and/or the Director) and the Pathologist.

In our area we all turn out as soon as the alarm is sounded and the minimum work is done at the scene until we are all assembled. We can then work as a fully integrated team.

All should wear gloves (preferably leather or rubber) to obviate fingerprints and fibres. Care must be taken where feet are placed because of footprints. Smoking is not allowed so that matches, cigarette ends or ash are not deposited. Only the minimum number of people is permitted near the body.

' Photograph, look and touch last ' should be the motto.

The greatest attention must be paid to detail, and even the most minute object must not be overlooked.

Remember that:

1. The investigation of a scene is a destructive act and nothing can ever be replaced exactly as it was.

2. Removal of the body may cause things to drop off it or become attached to it.

3. Undressing the body has the same effect.
4. So does post-mortem examination, and prior washing.
5. Admission in the witness box of omissions renders one easier to discredit.

My scene bag contains all that (as I have learnt from experience) is necessary to collect and retain specimens. Tubes, bottles, envelopes, forceps, scissors, swabs, Pasteur pipettes, etc. All containers are best labelled in advance for ease of handling. A list is kept.. Cellotape strip is very useful for microscopic fibres and small hairs. Watch forceps may mark objects.

If a body is removed from the scene, make sure that it is moved on to virgin ground—preferably covered by a plastic sheet or some clean material. I watch the body put into the container for removal. Do not use blankets as they shed fibres liberally.

At the mortuary I remove each garment carefully and place it on a clean paper. Hanging garments up has advantages, but dust and fibres can fall off. The body is then carefully inspected and samples taken.

Glass, paint or bones, fibres, bullets, shot, wads, bits of weapon, dust in wounds or anything else found in the body are all carefully kept and recorded.

Control samples (head, pubic and axillary hairs, blood (for grouping), nail scrapings, swabs, stomach contents, urine) should all be labelled, listed and handed to the Forensic Laboratory Staff or Police Officers detailed for the purpose.

Now, why all this fuss? It is because of *contact traces,* a subject about which we meet to-day.

These are macroscopic or microscopic objects, fragments of objects or other material that associate the dead person with the weapon or agent involved in the death, that associate the weapon or agent with the suspect, associate the suspect with the scene and establish contact between the deceased and the scene. The place where the body is found may not be where death occurred.

Our task is to ascertain the truth to the best of our skill and knowledge. Patience is essential. It is often vital to establish that a crime was not committed to prevent an innocent person from being convicted. One in four of my major investigations turns out to be a crime. I do not refer to ordinary Coroner's post-mortem examinations). *An apparently simple post-mortem examination may turn out to be a case of murder. Remember further*:

1. You never know what may be of value in the subsequent proceedings.
2. Continuity—minimum of handling both of the body (identity) and samples.
3. It is necessary to prove each sample in the Courts.
4. The onus of proof is on the Prosecution.
5. Never before have Laws, Medicine and Science so co-operated.

Before I conclude and hand you over to the speakers who will give you the greater detail, I would just like to mention the examination of suspects.

I am occasionally called upon to do this in the absence of trained Police Surgeons—although it is really their duty.

I always obtain the permission of the individual in writing and confine my examination to the surface of the body, a search for contact traces and the collection from the suspect of appropriate samples, e.g. head hair, pubic hair, nail scrapings and blood.

I avoid psychiatric observations and, if in doubt about the individual's health, request the Police to obtain the appropriate medical advice.

Contact Traces

Prof. J. Glaister (University of Glasgow)

My contribution to this symposium on *Contact Traces* is to discuss the subject of hairs. I have been given easier tasks than to deal with such a detailed subject in the short span of 20 minutes, the period allotted to me. On consulting *The Concise Oxford Dictionary,* I think I am correct in asserting that you will find there, among others, two meanings of *symposium,* viz. ' any drinking-party ' or ' an ancient Greek after-dinner drinking-party with music, dancers or conversation.' If you accept these definitions then I trust you will not be disappointed with the *Proceedings* this morning.

I have always been deeply interested in the *Theory of Interchange* connected with criminal investigation and take the view that the association of hairs with weapons, clothing and other objects may assume high importance in forging a link in a chain of circumstantial evidence. It is hardly necessary, however, for me to add that in order to obtain such specimens, a meticulous examination of the scene of a crime becomes imperative. Such specimens (if recovered) must, of course, be identified with the act of finding and thereafter be labelled and preserved for subsequent detailed, laboratory examination.

Usually these examinations take the form of comparisons between the specimen or specimens found and the hair of a suspect or suspects. Such comparisons are anything but easy and should never be made lightly and superficially; for they demand careful preparation and painstaking microscopical scrutiny. Before attempting identification, an examiner should have a thorough general and detailed knowledge of hair characteristics and for the purpose of comparisons, have available a large collection of previously identified human and animal hairs. The collection should comprise not only mounted specimens in the longitudinal and transverse planes, but also photomicrographs to which reference can be made.

Such provisions are necessary before an examiner is justified in expressing an opinion as to the likely source of hairs of unknown origin. It was in 1919 that I first became deeply interested in the study of hairs. From the literature it seemed that the advice given, which was to the effect that ' a personal study of hairs will yield much practical and important information from the jurists' point of view,' was of little practical value unless such advice was put into practice. The existing Atlases of Lambert and Balt-

hazard, Friedenthal, Waldeyer and Grimm and Fritsch, although of considerable assistance in hair study, were largely composed of drawings, greatly enlarged photographs of racial characters of hairs. These did not appear to me to furnish the natural appearances of hairs seen in the course of routine laboratory microscopical examination. It seemed, therefore, that a useful purpose could and should be fulfilled by forming a comprehensive collection of hair specimens and their photomicrographs. With the kindly co-operation of many colleagues, hair-dressers and others, human hair was collected. Officials of zoological collections and museums, taxidermists, furriers and those in certain branches of industry supplied the animal hairs.

In all some 1,700 specimens were examined in both longitudinal and transverse planes and classified for future reference.

It will be obvious that in the very limited time at my disposal this morning it is impossible for me to discuss the findings in any detail; but I would say that the work was rewarding and the specimens collected have provided me with useful laboratory controls over the past 38 years. During this period I have examined large numbers of hair specimens in the role of traces relating to the *Theory of Interchange*, and the collection formed has proved invaluable.

These examinations have been associated with the following types of case, viz. murder, assault, rape, unlawful carnal knowledge, acts of indecency, sodomy, bestiality, removal of hair by cutting, fur thefts, faking in dog racing, cattle maiming, adulteration of animal foods, motor car and other vehicular cases. As the result of this varied experience, some useful points have emerged to which perhaps I might usefully direct your attention.

An important feature of hair lies in its retention of structural characters for extremely long periods. It has the capacity to resist putrefactive changes. For this reason hairs may prove significant in relation to identification of putrefied, mutilated or dismembered remains. I have examined hair from embalmed subjects classified as belonging to the pre-pre-Dynastic Era and from those of the 11th, 12th and 20th Dynasties. In these specimens the microscopical features were clearly seen even in transverse section. It is obvious that in the present-day approach to the investigation of crime, those who profess to provide scientific aids must have an intimate knowledge of the characters of hairs and the necessary technique for their examination, since such traces frequently figure as exhibits in a large variety of cases. From their detailed examination many important points may emerge to forge quite an important link in the chain of circumstantial evidence.

It is advisable for an examiner to submit, with his report, photomicrographs illustrative of the points on which he founds his opinion. Such a course tends to shorten subsequent examination in the witness-box. The

necessity for meticulous examination is reflected in the type of question which may be put to a witness. For example:

Is the hair that of a human being?
Has the hair been dyed?
Has the hair fallen out, been pulled out or severed by cutting?
If severed, was a sharp or blunt instrument used?
Is this an animal hair?
If so, to what class of animal does it belong?

These are only a few of the questions which may be asked.

It must not be overlooked that the most important information for identification purposes can be derived from the examination of cross-sections and that it is quite feasible to section a single hair by the employment of the proper technique. The sectioning of hairs is often a difficult and delicate manoeuvre fraught with many obstacles and demanding both patience and perseverance. The task is not lightened when only a few specimens or a single specimen is available.

In the examination of cross-sections of a hair bundle, the examiner must remember that the hairs have been cut at different levels and varying angles. In his interpretation of their microscopical appearances due regard must be given to these factors. It is therefore necessary for him to select a number of specimens which show the predominant characters for special examination and ultimate assessment.

In denoting sex, the length of head hair can prove important, but the peculiarity of both male and female in this respect frequently undermines reliability of opinion on this basis. We know that the hair from aged persons is usually white or grey in colour, but it is a wise precaution to refrain from expressing an opinion that a hair specimen is that of an old subject merely on account of its colour or microscopical appearance of degeneration, since relatively young subjects sometimes become prematurely white or may show an odd grey or white hair in an otherwise well coloured mass of head hair.

Occasionally hairs may show irregularities in the contour of the shafts, due to permanent waving or to abnormality. Such findings may have a significant reference to identification. In some cases, debris adherent to human hairs may prove of value in suggesting the probable site from which hairs have come. For example seminal fluid, faecal material, vaginal or nasal secretion, food or cerumen give indications. It is therefore expedient to make a preliminary microscopical examination before cleansing the specimens.

To determine the source of origin of hair is usually a difficult, tedious and prolonged affair. Even after the most exacting examination, exercise of caution is required in the expression of the resultant opinion. It is not, of course, possible for any examiner to state that a given hair specimen of unknown origin belonged to a certain person, but it is sometimes possible,

when a sufficiency of hairs is available, to state that they did not belong to a certain individual. The diversity in the appearance of human hair is very great, on account of the many shades and characters it may assume, the alterations due to age, and the different parts of the body on which it has grown. Even in the same individual the hairs exhibit wide divergence in microscopical appearances. There is a tendency for some examiners to express over-didactic opinions regarding comparisons of hair samples. The opinion that 'specimen A is identical with specimen B' should never be expressed. It is an unwarranted opinion, since human hair specimens are never identical. It is only by an assessment of the dominant gross and detailed characters that comparison can be made and such comparisons can never fall within the category of, for example, finger-print comparisons, so far as accuracy is concerned. Following detailed examination of two specimens, aided when necessary by adequate controls, the furthest length to which an examiner is justified in going when expressing an opinion is to state that:

> As the result of the foregoing examination, I am of the opinion that comparison of hairs of specimen A with those of specimen B showed that the gross and detailed characters were such as to be consistent (or inconsistent) with a common source of origin.

He should never be inveigled into a definite answer as to whether a hair examined belonged to a given person.

In conclusion, I would like to add that a considerable amount of work on hairs still remains to be done, particularly in relation to the retention of certain toxic substances—a further important hair characteristic.

During the past two years, in my Department, largely under the guidance of Dr. Rentoul, Mr. Hamilton Smith has been engaged in research in connexion with the detection and quantitative estimation of such substances, particularly arsenic and antimony. The work, which has been specially directed to the harnessing of modern activation techniques for such purposes, was promoted by the Medical Research Council and the University of Glasgow, which arranged for the co-operation of the Isotope Division of the Atomic Research Establishment at Harwell and the Western Regional Physics Department.

For the purpose of this paper, the most interesting part of the work has been the discovery of a quick and accurate technique for the estimation of arsenic in biological materials, notably hairs. Single hairs have been successfully analysed and it has been found that after oral arsenic administration, radio-activity, in varying intensity, dependent upon dosage, is detectable in the part of the hair of the head into which excretion has occurred. Single hairs weighing as little as 0.5 milligramme have been used successfully. This research, although still incomplete, has already shown itself useful in practice.

An elderly subject repeatedly became ill after food with vomiting and abdominal pain. She was admitted to hospital and a laparotomy was performed but nothing abnormal was found. She continued to take food without incident during her subsequent stay in hospital. Returning home, however, the patient had a recurrence of the illness and criminal arsenical poisoning was suspected. Samples of vomitus were submitted for chemical examination by conventional methods. The analyst reported finding traces of arsenic. The clinical history supplemented by the analytical findings caused apprehension in the minds of the investigators. Suspicion became focussed on an attendant relative and on this account we were approached by a somewhat worried Procurator-Fiscal. Within a few days we were able to make an activation analysis of hairs and to demonstrate that the arsenic levels were well within normal limits. As a result, several persons were cleared of suspicion and police enquiries discontinued.

We are hopeful that when the work has reached conclusion some useful practical information may be disclosed.

I have deliberately omitted reference to laboratory techniques because I felt that little purpose would be served in dealing theoretically with such a practical subject which can only be studied within the laboratory. I hope, however, despite this omission, that I have stimulated some of my listeners to further research in this interesting branch of Forensic Medicine.

Contact Traces: Blood

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Blood and crimes of violence are so closely associated that they almost constitute a *sine qua non*. It may be, therefore, of the greatest importance to be able to identify the criminal or his victim from blood found at the scene of a crime.

When the Devil made Dr. Faustus use his blood to sign their eternal contract he demanded an inescapable stamp of individuality. As knowledge of the multiple factors involved in the structure of the blood grows, it becomes more and more clear that ultimately it should be possible to identify any individual by his blood.

Not only has the blood an inherently complex structure, with a variety of cells and a range of proteins, but also as the life stream of the body, delivering oxygen and nutrients to every cell and carrying waste products to be excreted by the liver, lungs or kidneys, it is the mirror of every bodily activity. Thus in suitable circumstances the sugar or alcohol content, the bilirubin level or the percentage of carboxyhaemoglobin can tell much of the victim's last hours and may provide important links in the detection of crime.

From the terms of reference for this Symposium I must temper my remarks so that as far as possible they apply to traces of blood and the difficulties inherent in their examination. I do not propose to discuss the detection of blood as such, but rather to assume that this has been established. I feel, however, that I cannot pass the spectroscopic examination of the blood without mentioning the possible use of an automatic spectrophotometer for scanning the range of wave lengths involved in the detection of blood products. This produces a photographic record of the absorption bands and, for example, allows the detection and estimation of very small amounts of carboxy- or sulphhaemoglobin.

Biochemical research into the breakdown products of haemoglobin has also opened up fruitful fields for forensic study. The curiosity of the schoolboy watching his purple bruise change its colour through blues to green and yellow has been replaced by the exact investigations of the protein and

porphyrin chemists, supported by their scaffolding of chemical formulae. These researches should offer much information regarding the chemical changes associated with the ageing of blood under varying conditions. These include degradation of the globin, the protein mass which forms the bulk of the haemoglobin molecule and bears the 4 specific oxygen carrying haem structures on its surface, and the accompanying steps leading to the splitting open of the haem ring and the eventual formation of bilirubin.

The globulin molecule has a further interest to our subject for subtle inherited peculiarities in its structure, although relatively rare are easily detectable and provide an excellent illustration of the principles of inheritance of individual characteristics. The molecule is formed from 600 or more amino acid units, or peptides, built from 19 different amino acids, and it only requires minor variations in their arrangement to produce a number of different forms of haemoglobin. It seems from recent work that the order in which the amino acids are combined is ultimately determined by the structure of the deoxyribonucleic acid, the chromatin substance of the nucleus of the haemoglobin forming red cell precursors. Thus the inheritance of the abnormal S haemoglobin, for example, is synonymous with the inheritance of unusual deoxyribonucleic acid in one chromosome of an autosomal pair, the peculiarity being in the arrangement of the purines and primidines forming the core of the double helical deoxyribonucleic acid molecule. For ease we speak of this deoxyribonucleic acid as a gene and, whatever the underlying physical chemistry, it is still the practice in genetics to consider that the inheritance of a characteristic is determined by the appropriate gene, or its variant the so-called allele, lying at a definite locus on one of a pair of chromosomes.

These genes are orientated in a definite manner along the chromosome and normally remain thus through repeated cell divisions. At the reduction division, which precedes the formation of a zygote, either an ovum or a spermatozoa, one chromosome from each of the 23 pairs is lost. On fertilization the chromosome pairs are reformed, with one member of each pair coming from the father and the other provided by the mother.

When like genes lie in the associated loci on a pair of chromosomes the individual is homozygous, i.e. all the zygotes must carry the gene; but when unlike genes (or alleles) are associated, the zygotes differ and the individual is called heterozygous. Each of the pairs of genes has to be considered separately. Some genes, such as that determining blood group A, are capable of expressing themselves when only present in one such locus; others, including the group O pair, require to be represented on both chromosomes to determine a characteristic. Many characteristics, such as the Rhesus blood group, have a more complicated inheritance and are dependent not upon a pair of genes, but upon several closely associated pairs.

Characteristics determined by genes on the same chromosome are said to be linked and can be carried through several generations, while characteristics determined by genes on different pairs of chromosomes are only associated by chance. Such chance associations occur relatively frequently with common genes, but family studies show that these genes do separate and are therefore said to segregate independently.

Only very rarely is there any departure from these principles of inheritance. Occasionally during the formation of a zygote a pair of chromosomes fuse cross-wise at the commencement of the reduction division and, when they separate, segments of the chromosomes, usually the ends, are found to have been exchanged. This phenomenon (called a cross-over) explains certain peculiarities of inheritance where linked characteristics eventually become independent. Genes placed close together seldom separate by a cross-over; while genes (and their alleles) occupying the same loci cannot be separated in this manner and changes in them can only occur by an alteration in their deoxyribonucleic acid structure known as a mutation. In normal circumstances such mutations occur only very rarely in Man, about once in every 50,000 gene generations; but the rate may be enhanced by physical and chemical agencies such as heat, X-rays and nitrogen mustards.

These then are the factors which underlie the characteristics which can be made to reveal the ownership of a drop of blood. Up to the present the chemist and the geneticist are still widely separated, both in the terms they use and in actual knowledge, but their fields approach as the immensely complicated nuclear structure is steadily unravelled.

The technique of electrophoresis has been of great value to the chemists. It has simplified the study of proteins, including the globin fraction of the haemoglobin molecule and, as only a small amount of material is required, it can be applied to trivial quantities of blood. The method makes use of the fact that if an electric current passes through a strip of blotting paper moistened in a suitable buffer solution it carries with it any electrically charged protein material, such as haemoglobin, placed upon the paper. The relative distance to which the protein is carried depends upon the sum of the charges on its constituent amino acids and various proteins may be separated from a mixture in this way.

Certain abnormalities of the haemoglobin structure can be identified by changes in the migrating distance on electrophoresis. Although rare in this country, such haemoglobins might play a part in the identification of a particular individual. Normal adult haemoglobin is determined by the presence of an A gene, not related to the blood group gene, on each chromosome and may therefore be called haemoglobin AA. In its globin there is a balanced pair of negatively charged glutamic acid groups and as a result it will travel much further at a pH of 8.3 than the haemoglobin of the sickle cell disorder, haemoglobin SS, in which an inert amino acid,

valine, takes the place of one of the glutamic acids. Haemoglobin CC, with a positively charged lysine group replacing one and neutralizing the remaining glutamic acid, hardly moves at all. The electrophoretic method can also be used to identify a number of other haemoglobin variants D, E, F, G, I, J and K. The type of haemoglobin found in the foetus and for a time after birth, haemoglobin F, can be separated from haemoglobin A, if the pH of the buffer is lower, but it is easier to distinguish the two by the resistance of haemoglobin F to alkaline denaturation.

Disorders of haemoglobin formation exist in both the homozygous and the heterozygous forms. In the former the condition has been inherited from both parents; the A gene is replaced by the S on both chromosomes; and an abnormal gene will be passed to all children. In the heterozygous form the condition has been inherited from one parent; affects only one of the pair of chromosomes; and is only passed to half the individual's children. The disorders of the haemoglobin are peculiar in that in the heterozygous state two different forms of haemoglobin exist side by side in the same blood, e.g. A and S, and can be separated as distinct bands by electrophoresis.

Some of the haemoglobin variants are relatively common in certain peoples. Haemoglobins S and C, although excessively rare in white people, are present in the blood of from 2–12% of negroes, and the E form occurs almost exclusively in inhabitants of Thailand. In addition to these, other variants of haemoglobin probably remain to be discovered, as the methods of identification are elaborated further, for it is believed that there must be a slight difference in the proteins of each individual.

A possible difficulty in applying the electrophoretic technique to small quantities of whole blood is that in the serum there are protein fractions, known as the haptoglobins, which might produce extra bands for they have the power to bind and carry traces of haemoglobin with them. These haptoglobins are themselves of an inherited nature and by their electrophoretic mobilities divide individuals into three types; group (1–1) with two haemoglobin carrying bands; group (2–2) with three; and group (2–1) with four.

Perhaps the most important features in relation to the inheritance of the blood are those which mark it as human as opposed to animal. Blood from the lower genera may be distinguished by the presence of nucleated red cells and these may be recovered from traces by the techniques to be discussed in relation to the white cells of the blood. With blood from different mammalian species, while the leucocytes may be distinctive, usually either a precipitin or an antiglobulin inhibition test is required. The ordinary precipitin test can be made more sensitive by the introduction of particulate matter to show up the flocculation, or by a modification such as the use of the agar gel diffusion plate. In the latter technique the test

and control materials are placed in equidistant holes in the gel and diffuse towards one another. If a reaction occurs between any of the reagents, a white cloudy line will develop in the gel where they meet. The anti-globulin inhibition test depends upon the power of human globulin to inhibit the agglutination of human cells, sensitized with a suitable incomplete antibody, by a specific antiglobulin serum and is an extension of the Race-Coombs technique. The test is highly sensitive and, for example, by this means it is possible to follow for several days the fate of as little as 0.5 ml. of a goat serum injected into a rabbit's circulation.

The human blood groups, inherited in accordance with the principles just defined, are of the highest importance in the determination of individuality. Identical twins have the same type of blood and close relatives may have certain similarities, but with over a million known variations the chance of others having identical blood is small. Thus blood grouping, embracing all the available systems, can select an individual from a group containing no blood relation, with nearly complete certainty. It must be admitted at the outset, however, that complete genotyping of an individual's blood is a highly skilled task, requiring rare sera and much more than a trace of reasonably fresh blood.

Nine major blood groups are known and about the same number of lesser groups, some of which may eventually be tied into the major systems when more is known about them. The inheritance of the antigens of the main groups is now well established. The gene and its alleles, or the small group of associated genes, for each group, are located on a different pair of chromosomes and therefore the groups segregate separately.

Mutations and cross-overs explain certain breaks in the inheritance of the blood groups which have been recorded, but there is also the possibility of an individual inheriting the rare 'Bombay' gene which suppresses the ABO antigens. Such an individual appears to be of group O, but can pass the A or B antigens to offspring. Equally rarely a double blood group occurs in a chimera, the individual's own group, which is transmissible, and the non-transmissible, possibly dominant group grafted through the common placenta from a dissimilar twin.

Fisher has arranged the major groups in order of their ability to distinguish between two samples of English blood as follows:

System	*Percentage Failure*
MNSs	16.4
Rh (CDE)	19.5
A_1A_2BO	32.8
Kidd	37.5
Duffy	38.3
Lewis	56.7
P_1P_2p	66.6
Kell	83.7
Lutheran	85.9

The minor antigen-antibody blood group systems include a 'general group,' a 'private group' and a 'race group.' The 'general group' antigen is present in the cells of almost all individuals, at least in any one geographical area. In this country only 1 person in 2,500 lacks the Val and only 1 in 250 the Yt^a antigen. In contrast the private groups, Levay, Jobbins, Becker, Ven, Wr^a, Be^a, Ca, Rm and By, are apparently limited to single families and appear to have arisen as recent mutations. The other minor systems are restricted to certain racial divisions for example the Di^a antigen, which occurs in up to 36% of South American Indians.

The antigens characteristic of these groups are situated in the stroma of the red cells, not just as a general constituent of the protein structure, but apparently in a definite location in the cell. Some placed on the surface are easily demonstrated, others lying deeper in the cell substance are revealed after using proteolytic enzymes, such as trypsin or papain, to open up the surface.

The ABO and related H antigens are not limited to the red cell stroma, but are found in the tissues and also, in some 75% of individuals, in the secretions. Such persons possess an inherited, Mendelian dominant, secretor factor which determines the secretion of the appropriate antigens in their saliva, seminal and other body fluids. Substances closely resembling, if not identical with the blood group antigens, particularly the ABO factors, are widely distributed in nature; in animals, plants and bacteria and it has even been suggested that the so-called natural antibodies are acquired through repeated contacts with these antigens from earliest infancy.

The ABO system is that most used for the purposes of identification, mainly because of the technical simplicity of the methods required. It consists of the antigens A_1, A_2, B and O, determined by a single pair of genes and their alleles. The possible genotypes, in order of their frequency in the south of England, are shown in Table 1.

Group O Genotype	*A_1*	*A_2*	*B*	*A_1B*	*A_2B*	*Frequency*
00						44%
	A_1O					28%
		A_2O				9%
			BO			8%
	A_1A_1					4%
	A_1A_2					3%
				A_1B		2%
					A_2B	1%
		A_2A_2				·5%
			BB			·4%
44%	35%	9·5%	8·5%	2%	1%	100%

A natural antibody, specific for any antigen missing from an individual's cells, is present in the serum from early childhood. Thus A individuals have always the antibody anti-B, B individuals anti-A and O individuals

both anti-A and anti-B in their sera, while A_1B individuals have neither. In addition many A_2 and A_2B individuals have a pure anti-A_1 antibody, which only agglutinates A_1 cells, in contrast to the anti-A in group O blood which agglutinates both A_1 and A_2 cells. The rare antibodies anti-O and anti-H, which react with both O and A_2 cells, can be found in Man, but are more frequently obtained from animal and plant sources.

If only a trace of blood is available, the red cells are likely to have broken down as a result of drying and are therefore not available for direct testing for the antigens. Antibodies, however, may be extracted quite readily in a little distilled water or saline, care being taken not to dilute them too much as they may not be very strong. Such an extract should be tested at room temperature against A, B and preferably also O cells, with positive and negative control tests using similar extracts of the material upon which the stain was found, to exclude anti-A or anti-B substances of extraneous origin.

Although much more difficult, it may be possible to demonstrate the group antigens in the remains of the red cells by their power to absorb the antibody from a test serum. The antibody to be used is carefully titred with known cells. A dilution, probably four times the end strength is chosen and an equal amount of this is mixed with an extract from the stain containing the cell debris. After a suitable interval the solid material is spun down, the fluid removed, serially diluted and tested for residual antibody with appropriate cells. Controls, including a similar extract from the material bearing the stain, must be set up at the same time. In such tests the quantitative relationship between the antigen and antibody must be borne in mind and adequate quantities of red cell stroma used to absorb the antibody.

By special techniques ABO antigens may also be demonstrated in various pieces of tissue found at the scene of a crime. With secretors too the antigens are present in any proteinous fluid including saliva, ascitic and seminal fluids and may be detected by their ability to remove the specific antibody from a serum so that it can no longer agglutinate the appropriate cells. Davidsohn and his collaborators in the Commitee on Medico-legal problems of the American Medical Association mention the detection of these group substances even on cigarette stubs and postage stamps.

The rarity of other group antibodies is usually sufficient to exclude them, but if there was any reason to suspect their presence and rather more than a trace of blood was available, extracts could be tested against a panel of cells covering, both positively and negatively, the antigens of the main groups. Such cells, obtained from selected cell donors, are available in laboratories accustomed to performing genotyping and can be stored frozen in glycerol over long periods. To detect possible antibodies the tests would have to be carried out at room temperature and at 37° C. and

by the antiglobulin technique. Once again controls from the material upon which the blood was found would be essential.

If adequate numbers of cells in a good state of preservation could be recovered, and this admittedly requires more than a trace of relatively fresh blood, the antigens of the other blood group systems could also be tested for directly. With rather less well-preserved material it may be possible to apply some of the appropriate absorption tests.

The antigens of the MNSs system are useful as they are evenly distributed and therefore divide the population into a number of even lots (Table 2):

Antigens	*Frequency*
MNS	27%
MsNs	23%
MMS	21%
NsNs	15%
MsMs	8%
NNS	7%

The related Hu and He antigens although found in 23 and 3% of West Africans respectively are almost absent from Europeans. Antibodies in the MNSs system occur infrequently in humans and are usually derived from animal sources.

The P system is less useful. P_1 predominates and occurs in 79% of English people, P_2 in 21% and p very rarely. Natural antibodies occur particularly in p individuals.

The complicated Rhesus system is perhaps the best understood, after the ABO system, as there has been much interest in the part it plays in the development of erythroblastosis foetalis, now better known as haemolytic anaemia of the newborn. This blood group system depends upon 5 separate factors determined by a set of closely related genes and their alleles:

D with D^u and d; C with C^w, C^u, C^x, c and c^v; E with E^w, E^u and e; F and f; V and v.

Antibodies are known to most of these, but only 4 are commonly available, viz. anti-D, anti-C, anti-E and anti-c. The groupings which can be separated with the 4 antibodies are:

CDe/cde	32%	
CDe/CDe	17%	
cde/cde	15%	Usually CDe and cDE are linked with
CDe/cDE	12%	Fv and cde with fv.
cDE/cde	11%	
Other groups	13%	

In certain circumstances anti-V, if available, would be useful for the V antigen is present in the blood cells of 40% of negroes but only in 0.5% of whites.

The Lutheran and Kell systems are not well distributed for identification in general. In the Lutheran system 92% of individuals are Lu a –ve and only 8% Lu a +ve, while with the Kell 91% are kk and only 9% are Kk. The latter system is important on account of the transfusion and pregnancy troubles caused by the antibody anti-K, created in kk individuals by contact with the K antigen either through a previous transfusion or as the result of a pregnancy with a Kell positive child.

The Lewis system is difficult as it seems to be connected with a serum antigen, which becomes attached to cells. The system is also linked with the ability to secrete ABO antigen; 72% of whites are Le (a–, b+) and are secretors; 22% are Le (a+, b–) and non-secretors; and secretors and non-secretors are equally divided among the 6% who are Le (a–, b–).

The Duffy system might also be useful in certain instances, for 65% of whites are Fy a (a+a+) and (a+b+) and 35% Fy b (b+b+), while 68% of negroes are Fy (a–, b–). Antibodies in this group are quite well known and arise as a result of transfusions or pregnancies.

The Kidd system is well distributed and if sera were available could be of use in distinguishing individuals. In England the frequencies are:

Jk (a +, b –)	27%
Jk (a +, b +)	51%
Jk (a –, b +)	22%

With full genotyping of the blood using the various systems mentioned the chance of identifying an individual would be quite good, particularly if there were some rare feature in the antigenic structure of the cells.

Other features of the blood, such as disorders of the clotting mechanism, including haemophilia, might at times provide features of interest in identification of an individual, but in general fresh samples of blood would be required for any of the specific tests.

In recent months we have been working on methods of recovering the leucocytes from blood smears on various objects. If a smear of blood upon a piece of glass or other clear material is available this can be stained directly and examined as a blood film. Usually the stain will be too thick, but this may be overcome by treating it by the thick smear technique employed in malarial surveys. For this purpose the red cells are lysed with water or dilute acetic acid before staining. Good white cell preparations can be obtained in this way, even with blood smears several months old and in successful preparations it may be possible to identify the sex nodules or 'drumsticks' characteristic of the neutrophil and eosinophil leucocytes of the female. The technique is, however, very laborious and must be improved before it could be used as a routine. This is particularly so when it is recalled that even in impeccable blood smear preparations from females on an average only one in over 30 neutrophils has a drumstick.

If no reasonable piece of glass is available, the task is much more difficult. So far only a few cells have been recovered from stains on cloth and none have had a drumstick. With smears upon opaque solids it is usually possible to transfer some of the blood to a microscope slide for staining and examination.

The technique is as follows. The smear is wetted with water, allowed to soften for a few minutes and then an attempt is made to transfer as much as possible to a glass slide, which may have been prepared by being covered with a very thin layer of celloidin or serum. The lysed blood is then stained and examined. The yield of cells recognizable as coming from the female is small and the possibility of a mixed blood is not excluded.

From this brief review of the possible applications of haematological methods to the investigation of small quantities of blood, such as might be found as contact traces, I hope I have indicated some fields which might be explored by those interested in this aspect of forensic medicine.

Summary

The possible methods of identifying an individual from traces of blood are reviewed. The blood groups are the most useful, but chemical changes, abnormalities of the haemoglobin and of the clotting mechanism and recognition of the sex drumstick in the leuocytes might be considered in particular cases.

Contact Traces

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Contact traces are ever with us. Their detection to-day appears to get more difficult and complicated.

The scene of crime to me as a policeman is a very sacred place and I know amongst you here there are many whom on occasion I have had the pleasure of accompanying. To those also it is a sacred place.

Unfortunately in our big metropolis of London we have these scenes coming to light by the answering of '999' calls, and one is often confronted at the scene of the crime by the foot impressions of, shall I say, 4 stout policemen who have transcended the normal avenue of approach and obliterated every mark made by the miscreant and others that the officers following might have been likely to find. This is a problem we have to contend with and it is only by lecturing policemen that this type of thing can be obviated. Of course, also in the policeman's mind must be the preservation of life or the apprehension of the suspect. Nevertheless, as clues are uncovered we do all we can.

A few years ago in a little place in the country a body was recovered from a ditch. The body bore wound markings which a pathologist said had been caused by impact with a moving motor car. The clothing of this person was taken to a Forensic Science Laboratory and swept with a vacuum cleaner.

These sweepings were treated in various ways. One of the things done was to treat the sweepings with acid and the remaining residue can be seen to consist of mainly a basis of silica and glass. By the use of polarized light one was able to differentiate the silica from the glass and the fragments remaining were spicules of glass which give similar characteristics of refractive index and specific gravity with glass which was found at the roadside a hundred yards from where the body was discovered. These larger fragments of glass at the roadside consisted of portions of a motor car headlamp lens, and here we must turn to our police officer who, after prodigious enquiries, discovered that an American serviceman in a car had enquired at a certain garage and had had a dented wing straightened out and a smashed headlamp replaced. This knowledge did not come to light until 3 weeks after the supposed event.

The car, at a later stage of examination, showed signs of recent repair and removal of the headlamp glass disclosed at the back of the reflector small pieces of glass which agreed in their refractive index and specific gravity with the pieces already mentioned.

This example, I think, amply illustrates the smallness of the sample which can be examined and the care which such examination necessitates. Such particles of glass could hardly be seen but they yield physical results which were positive. These deposits are often found in wounds where the body comes in contact with a glass surface which becomes broken.

The car mentioned was equipped with a Revenue Licence. This Licence on examination under filtered ultra-violet rays disclosed a previous written entry which had obviously been erased. This illustrates a technique which lends itself to forensic science examination very well indeed.

Next I illustrate 2 small pieces of paint, one recovered from a wound inflicted on the head of an individual and the other a control sample taken from a painted piece of wood (the suspect weapon). It can be seen that the layer structure of the paint is well defined and, indeed, it would be impossible to get 2 matching with such agreement unless they emanated from the same source.

Whilst on the subject of contact traces one must emphasize that the best contact traces of all and the one that has the highest evidential value is a finger-print. Its evidential value is high due to the fact that the finger-print is exclusive to one person. It is attached to that person and cannot, normally, be detached. This precludes the necessity of proving personal possession.

The next illustration has been made available by kind permission of Dr. Keith Simpson and shows the finger nail of a deceased woman. Without doubt it reveals a good example of a contact trace. During the fight with her assailant this nail became broken and embedded in the broken portion are fibres presumably snatched from the assailant's clothing. Material of this description must be preserved and one feels that the general preservation of the hands or, indeed, any body extremities is well worth while.

Protective material of good quality can be found in the modern production of polythene. Bags suitably anchored over the extremities provide adequate protection against accidental contamination.

We have had a very good talk on hairs. Fibres are related and, as with hairs, characteristic detail can be detected. With the fibre, however, one can take the evidential value a little further, chiefly because fibres can have colours which cover a multiple range when compared with their controls. This reduces the possibility of obtaining a mixture of such fibres of some other source.

I illustrate some material which was recovered from the finger nails of a negro. It is representative of vegetable debris and was subsequently

identified as Indian hemp. There are glandular hairs with the blob of resin on the top. Other features present which help to identify the species are the presence of cystolithic hairs.

The top of the skull of a lady who was out one evening exercising her dog on a golf course, is demonstrated next. She was brutally attacked and there appeared at the scene to be superficial evidence of rape. The body was found the following morning and for some 2 months no arrest was made. At the autopsy the skull was stripped and adjacent to the hole on the outer table some green paint was adherent, the inference being that it had been transferred there from the weapon which inflicted the injuries.

This paint was analysed spectrographically and compared with the green paint on a tee-marker flag. Agreement in their structure was confirmed. The tee-marker was without doubt the weapon used to inflict the injuries. It was heavily blood stained and provided the best evidence of all. In the dried blood fragmentary impressions caused by the friction ridges of the hands were apparent. Such was the investigation that in that area the police finger- and palm-printed some 4,000 of the population and at a later stage the impressions on the tee-marker were identified as belonging to a young lad who was resident nearby. He subsequently admitted the crime.

I would at this stage emphasize the bugbear of the forensic scientist: it is the mortuary keeper's sponge. I next illustrate the impression of a tyre track across the arm of a person killed as the result of a hit and run motor accident. Such contact traces are highly sensitive to water and after destruction and before photography cannot be reclaimed. The eccentricities produced by tyres are often capable of being recorded and compared with original markings. If the characteristics presented are sufficiently unique they leave little doubt about their origin.

For other reasons also, the mortuary keeper should pause before utilizing his sponge with any intent. A mark on the skin surface surrounding a bullet hole may have been caused by impact of powder with the skin surface, its distribution and intensity indicating the proximity of firing. Occasions have arisen where these marks have been removed by the washing of skin surfaces and when it becomes necessary to determine the distance of firing, the material to work with is absent.

The famous case where this technique was queried happened in 1926. Ronald Chesney's mother received a bullet wound early one morning. This was either self-inflicted, accidental or had been caused by Chesney, the son, firing a shot at very close range. The woman's body was admitted into the local infirmary and at this stage any trace of powder or markings surrounding the entry hole were removed, without noting, later precluding the possibility of any opinion being formed about the proximity of a firearm to the body. In this case the late Sir Bernard Spilsbury and Robert Churchill gave evidence for the defence.

Another example is of the estimation of the distance of fire. In this case the deceased person was a metropolitan policeman, killed one night some years ago by being shot 3 times in the back, one of the shots travelling down the femoral artery and passing out of the leg into the opposite knee. Witnesses who heard the firing described a sequence of shots.

At the time of the incident the deceased man was wearing a mackintosh. Infra-red rays were used photographically to portray the bullet holes and the characteristic powder pattern can be seen surrounding the mark indicating nearness of firing. The interpretation of this incident would be that of the body moving away, the last bullet hole revealing no powder marking at all.

Another problem is the markings on bullets which have been caused by the lands or projections in the barrels of the firearms. Such striations have often given a positive identification and confirm the firearm which discharged the shot. The comparison microscope is used for this type of contact trace.

Next I illustrate 2 bullets, one taken from the deceased person's body as found at the scene of the crime and the other fired as a control from the suspect weapon in the laboratory. The two when compared under a microscope reveal structure which shows quite definitely that they were both derived from the same firearm.

Another development destined to assist is when the comparison microscope is used. It is always apparent, when dealing with metal surfaces, either lead, nickel or copper, that one has to contend with a considerable amount of specular scatter due to the configurations of the surface. Such scatter cannot be controlled by normal methods such as polarization. It has been found, however, that by bombarding the surface of the metal in question with magnesium smoke the light scatter is subdued, the gross striations being accentuated and the subject is generally more suitable for the purpose of comparison.

The striations on a bullet may be obliterated by an over-enthusiastic gentleman probing for it—I am happy to say that in this case it was a surgeon endeavouring to save life and not a pathologist.

Here is a small piece of metal removed by a pathologist from the head of a deceased man. He had been severely battered with some heavy instrument. At a later stage police enquiries terminated at a factory. The small piece of metal was identified as swarf from a lathe. A man in the factory became suspect and from his lathe; control cuttings were taken. Subsequent comparison of the striations on the surface of the swarf showed agreement which left no doubt as to both having been made on the same lathe.

A few years ago, during the war, a skeleton was found in the basement of a bombed-out building. There was no clue to the identification of the

remains but a subsequent search in the vicinity yielded 3 keys and some documentary matter which was in an advanced state of decomposition. It was dried and put together. I illustrate the piece which was isolated and photographed using infra-red rays. A name was revealed. This was circulated to the national Press and some half an hour after publication a woman telephoned to say that her husband, whose name agreed with that revealed, had walked out of the house some 3 years before. He was known to be suffering from varicose veins and she, the wife, had not seen him since. To clinch the identity she informed the police that he had been a barber and operated from a shop in the Edgware Road. The mortise keys found at the scene fitted the locks in the premises. A collar found at the scene bore a laundry mark which was traced back to this particular individual. Such material was sufficient to satisfy the Coroner about the identity of the deceased person.

I show next a photomicrograph of the vaginal debris taken from one of the victims remaining from the efforts of Mr. Christie. This victim was, we believe, his last and in consequence the first to be removed from the alcove where he stored his bodies. This lady had been missing for 3 weeks and from an examination of the vaginal debris the presence of human spermatozoa was confirmed. The interesting thing about these is that their tails are still present and that they are still in cluster formation.

The second woman to be removed from this haven of rest was an Irish prostitute named Maloney. The vaginal debris on microscopic examination in this case disclosed a lot of gubbins, epithelium and bacteria. Intermingled with these can be seen human spermatozoa, some still with their tails and in this case it was established that the body had been dead about 5 weeks.

The last lady to be recovered revealed vaginal debris disclosing the presence of human spermatozoa. Decomposition had taken its toll; only the heads were present and are scattered over the microscopic field. This woman had been missing for some 7 weeks and there was nothing to dispute this period as being the length of time dead.

These cases reveal an interesting sequence confirming that even weeks after death contact traces are still available.

Whilst dealing with the subject of spermatozoa it is interesting, perhaps, to relate a case of bestiality with a domestic fowl. The accused stated that the chicken he had wooed and afterwards had had intercourse with had been buried in a plot of ground some 3 weeks before. Armed with a pick and shovel the local constabulary unearthed the animal. Subsequent laboratory examination of the cloaca showed the presence of tattered human spermatozoa—remarkable example of the persistence of this type of contact trace.

Trichomonas vaginalis may sometimes be mistaken by the inexperienced eye as for a spermatozoon. It is a flagellate, but due to the fixing solution the flagella have become retracted. It is a good test slide to put under the microscope for the uninitiated. One says, 'Yes, it's sperm. No, it isn't. Yes it is, it must be. No.'

Material removed from the apices of the lungs may show the characteristic diatoms normally resident in pond or river water; this may tend to corroborate the positive findings of a pathologist at autopsy. This is becoming a standard technique when bodies are recovered from water.

This talk has been rather rushed. I have tried to give you some idea of possible contact traces that might be found on the cadaver or at the scene of crime and some idea, possibly, of the evidence which may be derived from them.

Legal Principles Appropriate to Trace Evidence and Cross-Examination of the Expert

Dr. F. E. Camps: After Supt. Salter has blinded us with Science, I pass over to Mr. Lawton.

Mr. F. Lawton, Q.C.: That perhaps is a most apt introduction to the few remarks I am going to make. I am going to take you away from Science into the realm of human affairs and give you a peep at Forensic Art as distinct from Forensic Science. The point was underlined, I think, by Dr. Price because he addressed you as colleagues of his on the same line of enquiry investigating crimes on behalf of the Prosecution and giving you advice based upon his experience about the mistakes which can be made. I do not address you as colleagues in the investigation of crime; you will forgive me saying so. As a defending advocate, I address you as potential ignoramuses, clumsy clots and, possibly, as potential perjurers. That I think illustrates my point of view and shows how different it is from that of Dr. Price. May I make my comments under two headings? First, a short statement of what I understand to be the legal principles appropriate to trace evidence; and, secondly, a peep at the forensic technique of those of us who think we have some experience in cross-examination of forensic experts such as yourselves. By doing that I may assist you to put up some sort of defence against us. After all I am going to be fed by you to-night and it is only fair that for a short time I should act as a 'copper's nark.'

So far as the evidence is concerned, there really is not a great deal to be said. As you know, all facts which tend to prove the matters which are in issue in the case, are admissible in evidence unless there is a specific rule in the law of evidence which says they are not to be admitted. In a vast majority of criminal cases there are two issues:

1. Has the crime been committed? Has Mary Smith been raped? Has John Smith's shop been broken into? Has Martins' Bank been blown up?

2. Has the defendant committed it?

In most cases the defending lawyers are not greatly concerned whether the crime has been committed—there usually is no doubt about that—but whether their client Bloggins has committed it. It follows that a great deal of your work is directed to an issue with which the defending lawyer is not concerned forensically. They are primarily concerned to show that Bloggins may not have been connected with the crime; and those of you who have had court experience (and most of you have had extensive court experience) know that that is the issue which is fought out in most cases, if there is a fight at all.

So far as the first issue is concerned I cannot think of any rules of law which is likely to affect restrictively any of your activities. I suppose there may be cases in which your researches would reveal that a defendant had been in prison before, or something of that sort. In such cases your evidence would have to be led very carefully by the prosecution; but by and large what you discover can be put before the court provided it is relevant. May I in passing just call attention to one rule, a very humane rule which had fairly recently been underlined in the House of Lords. The case related to a policeman. He was charged with shop-breaking on a specific occasion and evidence showed that he had been on night duty in a certain place where there had been a large number of crimes but there was not a shred of evidence connecting him with any particular case of shop-breaking other than the one with which he was charged. The House of Lords pointed out that when a Judge comes to consider what evidence he should admit he must weight the probative value of the evidence against the prejudicial effect of it, and if he comes to the conclusion that the probative value is out of proportion to the prejudicial value then he ought to use his discretion in favour of the defendant. The only forensic illustration I can think of it is this: you might be giving evidence, for example, to help establish that Bloggins had committed rape and in the course of doing so you might refer to findings which suggested that Bloggins was not only a ravisher but a bugger too. As the issue before the Court related to rape, you might be required by the Court to omit any findings which suggested anything else.

I do want to point out strongly that in our Courts all evidence has to be given orally by someone. Here lies the weakness in trace evidence. You as the experts merely give evidence of what you found on a particular specimen at a particular time. You go to the scene of the crime; you see something there; you give evidence of what you saw there; and you say what you deduced from it; that is all. How it got there in another matter. I do not think this problem arises very acutely with regard to the matters with which you as doctors are normally concerned. It arises usually with the sort of evidence which is given by the Forensic Scientist. He receives at his laboratory a pair of trousers; at the same time he is handed a little package which he is told is a sample of the packing material which was used in the construction of a safe which has been blown open. He examines the turn-up of the trousers and finds traces of some substance there. He examines it chemically and microscopically. Then he examines the contents of the package which in appearance and other characteristics are identical with what is in the turn-ups of the trousers, but that is all he can say. The problem forensically is this: how did the debris in the turn-ups of the trousers get there? Some of us are very concerned indeed about the handling of garments and like objects between the time they are taken from

suspects and handed over to the Police Laboratory or to you, as the case may be. I have been dealing with the criminal classes long enough to know that you should never believe a word they say and, no matter how circumstantial their stories may be, you should always be sceptical. We defending lawyers do hear from time to time rather disturbing stories about trace evidence, of the sort of story in which a police officer comes in the cell and says, 'Here chum, let's look at your trousers.' The trousers are taken away and later thrown back in the cell and the prisoner is told to put them back on again. Shortly afterwards they are taken away from him again. Finally they are produced in Court and sure enough there is evidence from the forensic laboratory that debris has been found in the turn-up identical with the packing of the safe which the prisoner is alleged to have blown. This is a facet of the use of trace evidence which is worrying.

Now to the forensic technique of defending lawyers. There are two facets. The first is the action which is taken by the defendant himself. The criminal classes know well enough that by and large not much good is going to come to them by challenging the factual basis of your evidence. If you testify that there is a hair on the knife, unless it can be suggested that it got there in some odd way as, for example, by being put there by a dishonest policeman or something of that kind, most experienced criminals nowadays start off on the assumption that their defence has got to be built around the fact that there was hair on the knife and the problem they are faced with is how to explain the hair away. That is why Dr. Price's insistence on the proper guarding of the scene of the crime and of the exhibits is so very important. It is particularly important in finger-print cases because criminals know that it is not much good trying to challenge finger-print evidence factually. What they have to do is to say, 'Yes, that is my finger-print. I will tell you how it got there.' The favourite story to-day is this: when a weapon is found on their premises or at the scene of the crime and they are apprehended, they often allege that they were taken into the C.I.D. room and saw the object, knife or glass, etc. lying on a desk and a dishonest detective said, 'Just pick that up for a moment chum and have a look at it.' This explains the finger-print. From a practical point of view it is very important that police officers should safeguard the exhibits in such a way as to make it impossible for defendants to get hold of them at any stage. I am sure they do so to the best of their ability. In addition they should take precautions to see that if a lying story is put up, then evidence can be called to say that it could not possibly be true.

The avoidance of scientific and medical evidence by accepting the facts and explaining them away is met quite frequently. I had such a case a short time ago. I prosecuted a sailor for raping a girl down at Harwich. In view of his story that he had done nothing of the sort and had said

nothing more than goodnight to her, the prosecution called evidence to show that when he was arrested, which was shortly after the alleged rape, there were several stains on his trousers. When I asked him in cross-examination to explain these stains away he looked at me in complete disgust and said, 'Seminal stains, of course there were, I am a sailor. What else is there to do at sea?'

Now may I come to another aspect of forensic technique. How do we, as practising Barristers, set about the task of examining the forensic medical experts? Most of us when young read Marjoribank's *Life of Sir Edward Marshall Hall*. We learn that he was a great expert on poisons and how he was able to cross-examine Sir Bernard Spilsbury and other forensic experts of the age, raising this theory and raising that theory. As we got older we learn a bit more about him and come to appreciate that he was a first class actor and that his 'expert' knowledge was all probably part of the act. One of the things one learns by practice in cross-examining witnesses like yourselves, is that you are experts in your particular branch of the knowledge and that a knowledge acquired from textbooks can make no impression on a witness who really is an expert in his own field. For example, when I was young I cross-examined a physician about his clinical findings and then confronted him with a well-known textbook. I thought I had him covered but his answer was, 'Oh, of course he is from such and such hospital, but our hospital has been able to establish that he was quite wrong on that point.' Now what answer is there to that? Any one who has acquired his forensic technique the hard way, does not challenge the expert on his own ground.

Well then where does the challenge come? There are two aspects of your evidence. First, the actual aspect—what you have actually seen and, secondly, the inferences which you draw from what you have seen. Occasionally, but only occasionally, the advocate is able to trip up the forensic expert on what he has seen or what he finds in the course of experiments. This seldom happens with pathologists. It does happen more frequently with the forensic scientist because, in some of the laboratories, particularly in those dealing with food rather than the laboratories dealing with crimes, there is a bad practice of allowing laboratory assistants to do part of the experiments and things do get mixed. I had a food and drug case a short time ago where the vitamin properties of some product was in issue and to my astonishment the presence of 10% alcohol in the product had been completely missed and so had the presence of some 8 or 9 vitamins. However, that kind of thing is unusual. When advocates do find that you have missed something, then they pounce on it and spend the rest of the trial trying to persuade the Judge and jury that what you missed was the most important thing in the whole case.

Much the best way to deal with technical and expert evidence is to tackle the inferences which the expert draws from his factual findings. By and large, experienced advocates will not challenge you with theories because, if you are a good expert, there is unlikely to be an expert on the other side who will go into the witness box and support an opposing theory. But it is surprising in practice how doctors will stretch their inference a little bit too far. The art of advocacy is to encourage them to do so. When you are being cross-examined upon the inferences you have drawn, keep a look-out for the advocate who asks you whether you are going as far as this and then postulating some proposition to you. He is dangerous.

Finally, what is the value of forensic evidence? It is as valuable as the man who gives it. If he is one of the greatest scientists in the world, but is unconvincing in the witness box, then his evidence from a forensic point of view is valueless. Evidence, to have effect, must be given confidently and, above all, fairly. The witness who seems anxious to help the prosecution prove its case is a liability. The impressive witness is the one who does not seem to care about the outcome of the case.

Afternoon Session

Agonal Changes in Blood Serum

Professor W. Laves (Munich)

It is a great honour for me to have this opportunity of attending this meeting of your famous Society and of reading a paper.

I feel very indebted to you for the kind hospitality offered to my wife and myself, particularly so, perhaps, because I understand that this is the first time that you have had a guest from Germany.

The act of dying is not instantaneous except in the case of severe destruction of the brain or other vital organs. The shortest time is about 4 minutes, but it may last some hours. It is well known that oedema of the brain and lungs, contraction of the spleen and other changes due to severe hypoxia may occur.

My study is concerned with the question whether changes in the serum and plasma occur during the agonal period.

Unfortunately it is not possible to give you pathological details, since the examinations have been performed with a spectrophotometer. But I am sure you will agree that physico-chemical and physical methods are becoming more important in forensic medicine.

Some words at first on the material examined. This consists of blood samples from:

(*a*) Healthy adults.
(*b*) Persons injured in traffic accidents, before and shortly after death.
(*c*) Both ventricles, the femoral veins and so on of bodies within five hours of death.

Method

The specimens were centrifuged at 5,000 revolutions per minute for 10 minutes. Haemolysed samples were removed.

To 1 ml. of clear serum was added 3 ml. trichloro-acetic-acid. The mixture was then again centrifuged at 10,000 revolutions per minute.

The clear supernatant portion was examined using a Unican Ultraviolet Spectrophotometer between the wavelengths of 220–300 mμ, the cell used having a diameter of 1 cm.

Trichloro-acetic-acid (TCAA) extract of the blood serum of normal persons gives a uniform curve.

The curves represent the spectrophotometric behaviour of TCAA extracts of blood specimens taken from dead bodies. I have to mention that these

solutions had to be diluted 2 or 3 times with TCAA because of their very strong absorption.

These curves show a quite different form from that of normal sera. You will see that there is no minimum absorption at 250 mμ, and, in fact, a steep maximum absorption starts at 250 mμ reaching a peak at 260μ. A second slight maximum is sometimes visible at 285 mμ.

You will notice that the causes of death are quite different, for example:

Hanging;
Drowning;
Carbon monoxide poisoning;
Internal bleeding;
Fractures of the skull;
Coronary arteriosclerosis.

Having examined a small number of sera giving the same results, I formed the opinion that these findings might be a characteristic post-mortem behaviour of sera treated in this manner. However, as the numbers of cases increased other inferences appeared to develop.

The general form of the absorption curve may be the same, but there may also be a difference in the location of the maximum absorption (broader and reaching a peak at 265 mμ).

The causes of death in this series were:

Parathion poisoning;
Drowning;
Internal bleeding;
Fractured skull.

With still further examinations, it was found that there are further types of absorption curves. This group showed a broader or smaller peak at 270 mμ, the causes of death being:

Carbon monoxide poisoning;
Cyanide poisoning;
Rupture of the heart;
Fracture of the skull.

The last group shows a broad absorption beginning at 250 mμ and ending at 290 mμ with a peak at 280 mμ. The causes of death were fractured skull and internal bleeding.

From the various forms of curve obtained we are able to say:

1. There is a difference in the ultraviolet absorption curves of ante- and post-mortem blood samples, viz. a shifting of the maximum absorption from the longer to the shorter wave length.

2. This shifting is combined with an increased absorption. This means that the concentration of the absorbing material is much higher in the post-mortem than in the ante-mortem samples. Also, at a wave length of 260 mμ no absorbing material is present in the serum of live patients, whereas there is a high concentration in the post-mortem samples.

In order to ascertain whether these phenomena occurred during the agonal period or after death, rabbits were poisoned with CO or CO_2, samples of blood being taken 1–2 minutes before poisoning, during the stage of hypoxia and after death.

Ultraviolet spectroscopy gave similar curves to the human experiments and the shifting of the maximum absorption from 285 mμ to the region of 260 mμ took place during the agonal period.

We have also had the opportunity of examining cases of fractured spine given artificial respiration in an iron lung. Samples of blood were taken when the oxygen saturation was still normal, when paralysis began and shortly after death. Here again similar curves were obtained with shifting occurring during the agonal period.

It is thus possible to make the following conclusions:

1. Ante-mortem and post-mortem blood samples are different with respect to the ultraviolet behaviour of TCAA extracts of the sera.
2. These changes occur during the agonal period.
3. These findings are characteristic for normal healthy individuals.

We have examined the blood in cases of:

Heart failure;
Lung tumours;
Poliomyelitis with artificial respiration.

The shifting phenomena occurred where there was an oxygen saturation deficit of the blood and was intensified with an increasing degree of hypoxia, reaching its maximum during the agonal period.

The chemical changes accounting for this phenomenon are not yet completely understood. Haemolysis plays no part. Filter paper chromatography showed that organic phosphorus corresponds, for example, ADP and AMP are normally absent in the sera but often found in sera of cases suffering from hypoxemia and in post-mortem samples treated with TCAA. These substances give rise to a curve with a maximum absorption at 260 mμ. The other types of absorption curves make it probable that other substances, not yet identified, appear in hypoxaemia and the agonal period and further work is required to elucidate this.

Summary

1. A non-specific shifting of the ultra-violet absorption curve from a maximum at 285–260 mμ due to hypoxia or anoxia is described.

2. This observation might give us the possibility of discerning whether a blood sample has been taken before or after death.

3. The phenomenon occurs in normal individuals during the agonal period. Blood samples of persons suffering from hypoxaemia show the same phenomenon *intra vitam* but to a lesser degree.

4. The shifting phenomenon is due to hypoxia and anoxia.

5. It is possible that the concentration of the substances which are responsible for this might enable us to ascertain the length of agonal time. Further investigations are required to see if this is reliable or not.

Finally, my thanks to Dr. C. Corby for his help in preparing this paper.

Discussion after Professor Laves' Address

Dr. Enticknap: I would like to ask if Professor Laves has any quantitative data on the absorption that he gets at 260 and 285 mμ.

Prof. Laves: I have no quantative data, I have just the technique, which is always the same; but we do not know what is the cause of the maximum at 285 mμ. We know that the maximum at 260 is *post mortem* because it is not present in the human blood sera.

Dr. Enticknap: The reason I asked is that I have made a number of similar measurement at 270 mμ. These were obtained from control tubes during estimations of transaminase levels in post-mortem sera and they do show a rough correlation with the time elapsing between death and the collection of the specimen. Similarly, the opacity of the controls of phosphatase estimation on post-mortem sera is much greater than that of living sera. As both these findings can be explained by the accumulation of organic acids, such as oxalo-acetic acid and tyrosine, in post-mortem sera, in favour of which I also have some chromatographic evidence, I wonder if the changes observed by Professor Laves in the agonal period are due to the same substances.

Amniotic Fluid Embolism

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Amniotic fluid entering the maternal circulation has been held responsible for causing sudden death in labour and also for contributing to post-partum haemorrhage by interfering with the blood coagulation mechanism. The case to be described is one in which sudden death appeared to be attributable to amniotic fluid embolism and is presented not only because amniotic fluid embolism is a rare occurrence, but also because this case has certain unusual features which might have a bearing upon the possible mechanism of death.

Since the condition was first described by Steiner[6] and Lushbaugh[5] 64 cases have appeared in the literature, although only 4 of these in British journals. One report was made by Dennis, Goldie and Polson.[4]

Our patient was a married woman, 32 years old. In 1952 her first pregnancy was terminated as she was suffering from active Potts' disease of the spine. In 1955 she had a normal pregnancy and delivery. She became pregnant again in July 1957 and was perfectly well during pregnancy, except that from 33 weeks onwards it was considered that she had a moderate degree of hydramnios. At 38 weeks she was admitted to the Bristol Maternity Hospital, having noticed a few vague uterine contractions. She never became established in labour but continued to have occasional slight contractions. Nineteen hours after admission she had a small show of blood and mucus. Immediately thereafter a midwife palpated her abdomen and felt a very strong uterine contraction. About a minute later the patient suddenly became flushed with stertorous respiration. She was immediately given oxygen, but despite this she quickly became intensely cyanosed. Her respirations were irregular and ceased within a few minutes. Shortly afterwards her pulse had also disappeared.

On admission she had denied that her waters had broken and there had been no vaginal leak of amniotic fluid up to death. Fifteen minutes after death some liquor drained from the vagina, but was not measured or collected.

Autopsy Findings

The most prominent external abnormality was intense congestion of the face and neck and slight congestion of the limbs. The conjunctivae were oedematous and there were a few rather large subconjunctival petechiae and

ecchymoses over the upper half of both globes. There were petechiae in the mouth and the skin of the cheeks and fine petechiae over the front of the abdomen. Externally she presented a picture very like that of traumatic asphyxia. Internally all the veins and the right side of the heart were greatly dilated.

The uterus contained a foetus weighing 9 lb. 5 oz.; it was covered by a thick white layer of vernix caseosa. There was no meconium staining and the amniotic cavity was dry of liquor. The placenta was implanted on the anterior wall, its lower edge reaching to within 7 cm. of the external os. The membranes had ruptured and at autopsy were enveloping the shoulders but not the head. There was 3 cm. width of membrane between the edge of the placenta and the site of rupture of the membranes. There was a possible site of entry of amniotic fluid into the circulation at the lower edge of the placenta where it had separated from the uterine wall for about 1 cm. over a length of 5 cm.

The lungs were somewhat congested and slightly oedematous. The right upper lobe contained a few old tuberculous calcified nodules and there was fibrous obliteration of the pleural cavity. There were no other relevant abnormalities. The Pott's disease, which affected L2–4, had healed.

Histological Findings

In the haematoxylin and eosin preparations of the lungs a few eosinophil epithelial squames were seen in the vessels, after prolonged searching. In sections stained by the acid-picro-Mallory method and by the combined method described by Attwood,[1, 2] using alcian green and phloxine tartrazine, a few more squames could be seen, but they were still very scanty. There was no alcianophil material, no intra-vascular fibrin thrombi, and no foreign debris of any sort; no lanugo hairs were seen despite examination in polarized light. It was only when sections stained for fat were examined that any significant degree of embolism was demonstrated. In some of the arteries and arterioles there was sudanophil material which, in places, had the appearance of squames coated with fat. There were also some fine droplets of fat in the capillaries and mixed with the red cells in some of the arteries. Even so, the number of vessels containing emboli was not very great and could be compared, in quantity, with the mild degree recorded by most writers on fat embolism. There were no emboli in any of the systemic organs, except in a few of the uterine sinuses.

Blood Coagulability

The blood throughout the body was fluid at the autopsy 8 hours after death, and remained so after removal. No fibrinolysis could be demon-

strated in the post-mortem blood by the method of Biggs and Macfarlane.[3] Fibrinogen appeared to be completely absent. The presence of fluid blood in the absence of post-mortem fibrinolysis strongly suggested that fibrinogenopenia occurred in life. Fibrin emboli, as described by Tuller (1957) were not present here, and there was no evidence of where the fibrin had gone.

Discussion

Despite the paucity of solid amniotic debris demonstrable in the pulmonary vessels, this death must be regarded as being the result of amniotic fluid entering the maternal circulation. The sudden onset during labour of dyspnoea, cyanosis and venous engorgement is characteristic of these cases and no alternative explanation was found for it in this instance. Other workers have reported numerous control examinations of maternal deaths from known causes other than amniotic fluid embolism, and have failed to find even the smallest amount of amniotic debris as an incidental finding. The alteration of blood coagulability is strongly suggestive of the acute defirinogenation reported from amniotic fluid embolism.

There are several unusual features of this case:

Firstly, the occurrence before labour had become well established. Amniotic fluid usually enters the maternal circulation after labour has been in progress for a considerable time. A few cases like this have been described, including that of Dennis, Goldie and Polson,[4] in which death occurred without any signs of labour having commenced previously.

Secondly, there was only one severe contraction immediately before the onset of symptoms. It usually follows repeated severe contractions and has, for example, followed the administration of Pitocin.

Thirdly, that histologically the diagnosis could only be made easily from sections stained for fat. Although the presence of sudanophil material has commonly been reported, in only one case was it the main component. Again, that was the case described by Dennis, Goldie and Polson.[4]

Fourthly, this case shows the smallest amount of amniotic debris so far described.

The most probable explanation for the lack of solid amniotic material is that the amniotic fluid entered the circulation early in labour. Most of the previous cases reported have shown meconium staining of the liquor and much of the debris described would appear to be derived from the meconium. It has also been suggested that meconium in the amniotic fluid detaches vernix caseosa from the foetus.

The most important aspect of this case is in the consideration of the mechanism of death from amniotic fluid embolism. Three possible mechanisms of death have been postulated:

1. That death is due to pure mechanical blockage of vessels by amniotic debris. This is the most widely held view at the present time.
2. That blockage of some vessels causes a generalized pulmonary vascular spasm; and
3. That the entry of large amounts of liquor has an anaphylactoid effect. It has been suggested that this might be due to foetal blood group substances sometimes found in the amniotic fluid.

This case suggests that the mechanism of death is not a pure mechanical block, but that either pulmonary vascular spasm or anaphylactoid shock is responsible.

My thanks are due to J. Seymour Williams, Esq., H.M. Coroner, Bristol for permission to publish the pathological findings and to Mr. H. L. Shepherd for permission to use the clinical history of this case.

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Acute Renal Failure in Carbon Monoxide Poisoning

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Skeletal muscle necrosis leading to acute tubular necrosis of the kidneys is a rare complication of carbon monoxide poisoning, recognized almost exclusively by continental pathologists. In 1856 Klebs[1] described a 33-year-old man who had lain for several days in an atmosphere containing carbon monoxide. At autopsy 33 days later the right arm and thigh showed extensive areas of necrosis and the kidney tubules were said to be degenerative with a so-called 'interstitial nephritis' present.

In 1921 Gunther[2] reported fatal renal damage in a 49-year-old man who, after an alcoholic bout, had lain unconscious for about 11 hours on the cold floor of a room full of coal gas. He died one week later, the post-mortem findings being reported by Scharmann, who found waxy degeneration of the muscles of the left arm, thigh and leg. The kidneys showed pigment casts in the tubules and evidence of tubular damage.

Spühler and Zollinger[3] in 1941 described the findings in a 41-year-old man who, whilst drunk, exposed himself to coal gas with suicidal intent. Anuria followed and at autopsy kidney damage was evident with casts in the tubules. No muscle damage of significance was evident in this case.

In 1948 Hedinger[4] published a review of 30 cases of muscle necrosis in carbon monoxide poisoning, including 2 cases of his own.

Case 1: A 21-year-old girl who developed swelling of the right forearm followed by Volkmann's contracture of the fingers a month after exposure to coal gas. At operation the median nerve was found embedded in dense scar tissue within the flexor muscles.

Case 2: A 30-year-old man who developed a swelling of the right calf 10 days after exposure to carbon monoxide with subsequent calcifications of the muscle.

Skin lesions were described in 23 cases and a histological confirmation of muscle necrosis in 12 of the 18 fatal cases.

Sokownina[5] in 1954 reported the biochemical findings in a 31-year-old female who developed oliguria following exposure to coal gas. Treatment by intestinal perfusion with sodium sulphate, successfully overcame nitrogen retention which amounted to 340 mg.% N.P.N. on the 16th day of illness, returning to normal after 30 days. Muscle necrosis was not mentioned.

Mautner[6] published a case of attempted suicide with carbon monoxide, in whom temporary oliguria and raised N.P.N. (85 mg.%) was associated

with brawny oedema of the right shoulder and thorax with muscle damage demonstrable on biopsy. Mautner credits Baron Larrey,[7] Napoleon Bonaparte's surgeon, with the first description of muscle necrosis in carbon monoxide poisoning. This was in a group of French soldiers who, overcome by fumes from charcoal burners during the severe winter of 1812, developed extensive skin lesions and presumed muscle changes.

This paper reports the autopsy findings in 3 cases where acute tubular necrosis of the kidneys followed exposure to carbon monoxide. In the first case postural pressure led to an extensive area of muscle necrosis and subsequent renal failure; in the second patient muscle damage, whilst present, was not as marked; and in the third case muscle damage was confined to the superficial tissues beneath pressure sores. The last two cases suggest that anoxaemia *per se* plays a more important role in the development of the kidney lesion than muscle necrosis.

Case 1

A 23-year-old female Indian student was found unconscious in a gas-filled room, lying on her back adjacent to a small flueless gas radiator from which unlit gas was escaping. The position of the lower limbs, knees flexed and drawn up beneath her thighs suggested an initial squatting posture. Within a few minutes of discovery she had regained consciousness, being confused and one hour later on admission to hospital she was fully conscious, no COHb being found in a specimen of blood. Direct questioning revealed no history of suicidal intent, this accident having occurred due to her ignorance of the mechanism of gas heating. Feeling cold she had wrapped herself in a blanket, turned on the radiator tap, not realizing the necessity to ignite the gas. Hypotension was present out of proportion to the clinical state for 24 hours after admission to hospital and the following day urine volume was reduced but not accurately recorded. Three days after admission the blood urea level was 163 mg.%. Investigations to elucidate the cause of this nitrogen retention showed no evidence of anaemia, past renal disease, diabetes or exposure to known renal toxins, and gynaecological examination revealed no abnormality. At this time her general condition was good. Conservative measures for the treatment of acute renal failure were instituted and then 6 days after the accident an extensive soft tissue swelling with brawny oedema of the overlying skin was observed to involve the inner side of the right thigh, extending above the inguinal ligament, the limb being held in a position of external rotation. At this time the blood urea had risen to 325 mg.%, the bicarbonate level was reduced and serum potassium was 5.4 mEq./litre. During the following 3 days her general condition continued to be satisfactory but with the serum potassium rising to a 7 mEq./litre and the bicarbonate level falling

to 12 mEq./litre, haemodialysis was considered. Before it could be carried out she died quite suddenly, 9 days after the initial exposure to gas.

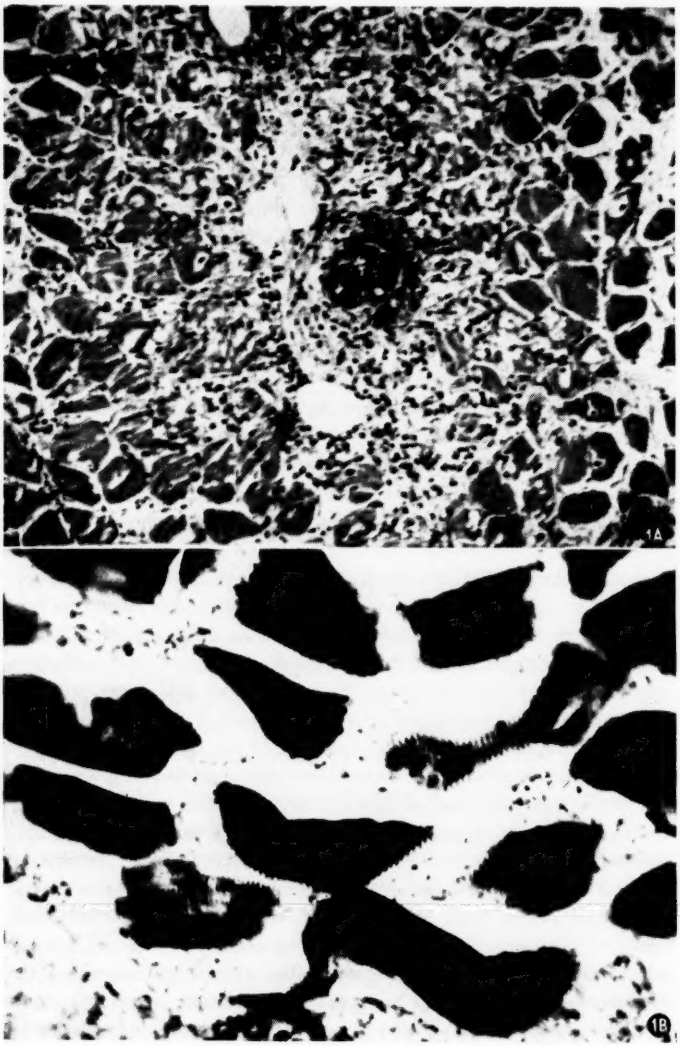

Fig. 1A. Section of adductor muscle showing disruption of fibres, arteriolar necrosis and cellular infiltration (H & E x 93).

Fig. 1B. Section of adductor muscle showing loss of striation (P.T.H. x 177).

Post-mortem Examination. At autopsy the upper third of the right thigh appeared swollen and incisions into it revealed oedema of the sub-cutaneous tissues. The entire underlying adductor muscle group for about 12 inches of its length was abnormally pale, yellowish grey and patchily mottled, some areas presenting 'fish-flesh' appearance. The musculature of the other limbs showed no abnormality, and with the exception of the kidneys the organs of the body showed congestive changes only. The uterus and adnexae were quite healthy. Both kidneys were pale and swollen, weight 210 and 220 g. respectively. The tense capsule stripped readily from the renal substance and the cortex was pale and moist and increased in width. There was no occlusion of the renal vessels.

Microscopical Findings. Sections from the affected muscle group revealed changes varying from loss of striation to complete degeneration of fibres (Fig. 1A). The majority of fibres showed a waxy appearance with fragmentation vacuolation and shrinkage of degenerate sarcoplasmic remnants. Some sarcolemmal sheathes were empty. Phagocytosis of dead muscle by histiocytes was evident and interstitial oedema with invasion by polymorphs, eosinophils and lymphocytes. No calcification was seen. Many arterioles showed necrosis of their walls and profuse cuffing with polymorphs and mononuclear cells which extended between the muscle fibres into the interstitial tissues (Fig. 1B). The larger vessels showed no abnormality. The term fibrinous vasculosis has been given (Lendrum[8]) to this condition of the arterioles which may be regarded as a result of exudation of material giving the staining reactions of fibrin into the vessel wall. The absence of generalized arterial disease and local disease of the femoral artery distinguishes the findings in this case from others where gangrene has followed exposure to carbon monoxide.

Sections of the kidney revealed glomeruli which were normal apart from the presence of an eosinophilic coagulum in some of the capsular spaces. The distal convoluted and collecting tubules contained dense benzidine positive casts. Whilst some of the distal tubules were intact, others were dilated and lined by a flattened regenerative epithelium (Fig. 2). There was marked oedema of the interstitial tissues with infiltration by chronic inflammatory cells and in the cortex evidence of a focal perivascular histocytic reaction adjacent to ruptured tubules. The blood vessels elsewhere in the body showed no abnormality.

Case 2

A 35-year-old woman was found unconscious on the kitchen floor of her home with gas escaping from the turned on taps of an adjacent gas cooker and a half empty bottle of sodium amytal beside her. She was discovered by her husband returning from work in the evening and he put

her to bed hoping that she would sleep it off. On the arrival of the wife's mother the following day, she was transferred to hospital where she was found to be deeply comatose with a poor peripheral circulation, the systolic blood pressure being 80 mm. Hg. During the next 24 hours little urine was passed and the blood urea was found to be 186 mg.% with potassium 6 mEq./litre and blood barbiturate level 0.5 mg.%. On the third day of admission the blood urea had risen to 300 mg.%. An examination of the blood and CSF sugar showed normal levels. Her general condition was deteriorating and the right foot became cold and pulseless. As the blood urea continued to rise to 128 mg.% haemodialysis was performed which resulted in a fall of the urea level to 128 mg.%

Fig. 2. Section of kidney showing tubular casts and regenerating epithelium (H & E x 158).

and potassium to 3.8 mEq./litre. Signs of bronchopneumonia were now evident and the patient died 7 days after initial exposure to coal gas.

Post-Mortem Examination. The upper part of the right leg appeared swollen and the adductor muscles on the right side showed patchy yellowish grey mottling for a distance of some 6 inches. The cerebral arteries were healthy but a small area of necrosis was seen in the globus pallidus. The heart was normal in size and showed no evidence of disease. A saddle-shaped adherent thrombus was present at the bifurcation of the aorta occluding the right common iliac artery but the origin of this clot was not apparent at the examination. The bronchial tree contained muco-purulent material and the lower lobes of both lungs showed a wide-spread consolidation due to confluent bronchopneumonia. Both kidneys were enlarged, the cortex being pale and swollen beneath a tense capsule, and a small area of recent infarction was seen at the upper pole of the right kidney as well as in the spleen. The uterus and adnexae were normal, there being no evidence of abortion.

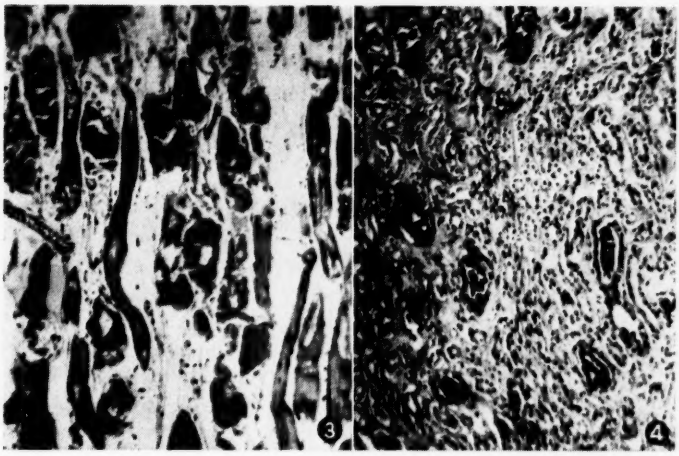

Fig. 3. Section of muscle fibres showing fragmentation and areas of striation (H & E x 80).

Fig. 4. Section of kidney showing pigment casts and tubules (H & E x 80).

Microscopical Appearances. Sections of the adductor muscle showed evidence of damage consisting of less of striation of the fibres, with small areas of fragmentation and vacuolation (Fig. 3). There was no evidence of arteriolar damage, the overall changes being much less marked than in Case 1. The kidneys showed evidence of an acute tubular necrosis with

very large numbers of pigment casts in the distal and collecting tubules and cortical tubular damage (Fig. 4. The infarcts were confirmed and the thrombus in the aorta was seen to be antemortem in structure.

Case 3

A 47-year-old man was found curled up unconscious in a cupboard of an empty house. It was not known how long he had been there, the discovery being made by chance due to the visit of a prospective house purchaser and his wife, their attention being drawn to a strong smell of coal gas in one room, the source of the gas being a fireside point. On his removal to hospital he was found to be deeply comatose with a pinkish colour and an unrecordable blood pressure. No COHb examination was done. The following day blood pressure was recorded as 120/80 mm. Hg., the limbs were spastic and the urine contained a large quantity of aspirin-like substances, the blood salicylate level being 17 mg.% Two days after admission the blood urea was 180 mg.%, urine output reduced and electrolytes otherwise normal. The only alteration in his condition until his death 12 days later was that coma became less deep and focal fits occurred in the left arm and leg. The last blood urea level was 296 mg.%.

Post-Mortem Examination. Pressure sores were apparent over the hips and shoulders with smaller areas over the sacrum, the muscle underlying these areas appeared healthy, but at the time of examination the general

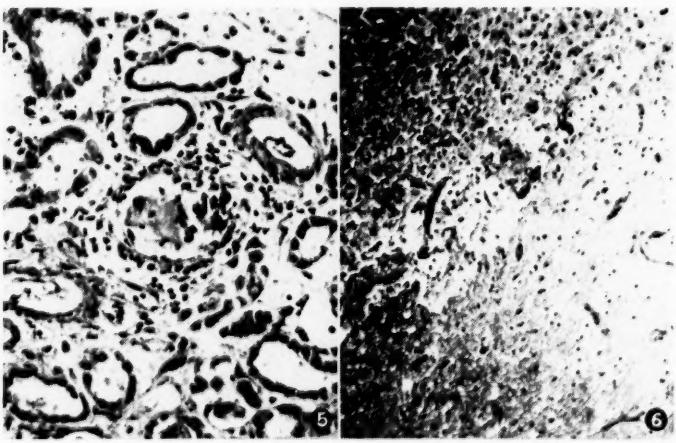

Fig. 5. Section of kidney showing tubular casts and interstitial reaction adjacent to ruptured tubule (H & E x 120).

Fig. 6. Section of brain showing well defined area of infarction (H & E x 80).

nourishment was very poor. Examination of the skeletal muscle elsewhere revealed no evidence of trauma or necrosis. Internally the arteries of the brain were healthy, with typical bilateral areas of softening in the globus pallidus. The heart was normal in size and the valves, aorta and coronary arteries quite healthy. The air passages contained some muco-purulent material and the lower parts of both lungs showed changes of hypostasis and congestion, there being no evidence of pulmonary disease. Both kidneys showed some pallor and swelling of the cortex, weight 200 and 205 g. respectively. The appearance of the kidneys suggested an acute tubular necrosis.

Microscopical Appearances. Sections of the kidneys showed typical benzidine casts in the distal and collecting tubules, oedema of the interstitial tissues, infiltration of chronic inflammatory cells and tubular damage including rupture (Fig. 5). Section of the brain and affected areas showed well demarcated cerebral softening with many fat-laden astrocytes (Fig. 6) and sections of muscle fibres beneath the marks of pressure showed areas of necrosis (Fig. 7).

Fig. 7. Section of muscle fibres showing necrosis (H & E x 16).

Discussion

The cases I have described and those from the literature I have quoted accord in their main features of exposure to carbon monoxide and subsequent renal failure. In many instances substantial muscle necrosis has been evident. It was certainly present in Case 1 but the significance of the muscle damage in Case 2 is doubtful and Case 3 lacked muscle damage of importance. In instances where muscle necrosis has been demonstrated, it is probable that myoglobinuria has occurred and as this may be a transitory phenomenon unless suspected it is likely to escape detection.

The association of myoglobinuria with acute tubular necrosis of the kidneys is well recognized. In Case 1 it seems probable that the combined factors of local ischaemia due to pressure and anoxaemia due to carbon monoxide were responsible for the muscle damage. This followed a 7-hour exposure to the gas, with presumed immobilization of the limbs, the damage being restricted to one lower limb. Pressure necrosis of muscle has been reported following periods of unconsciousness due to other agents. These include:

'Wine and sleeping tablets' (Jasinki and Brutsch:[9] A 43-year-old man who was found comatose after 2 days' exposure in woods developed swollen, painful legs and myoglobinuria with non-protein nitrogen of 252 mg.%);

Barbiturates (Touze and Thierry:[10] A 23-year-old female found comatose with the weight of the body resting on her right leg for a period of 16 hours, she developed renal impairment); and

Alcoholism and barbiturates. This latter group was reported by Fahlgren[11] who described 5 male alcoholics who developed myonecrosis following periods of coma and in two patients pressure of the muscle was cited as a possible causative factor.

Gordon and Newman[12] reported a case of a very obese 35-year-old man who was placed in a knee-chest position for laminectomy operation for 3 hours (a position reminiscent of the man in the cupboard, Case 2). The operation was abandoned and the patient subsequently developed oedematous calf muscles and died 7 days later in acute renal failure. Hence in some of the cases the assumption of an abnormal posture during immobilization has been evident.

The importance of decreased arterial oxygen and anoxaemia due to carbon monoxide has been mentioned; perhaps the gas interferes directly with oxidative enzymes of muscle by forming carboxymoglobin. It is known that myoglobin has 1/25th of the affinity of haemoglobin for carbon monoxide, so that it is apparent that myoglobin has 12 times as much affinity for carbon monoxide than it has for oxygen.

However, according to Hed,[13] postural pressure may influence the localization of the muscle injury in cases where it occurs even if anoxaemia plays a part in its development. Furthermore Adams, Denny-Brown and Pearson[14] have observed muscle necrosis in skeletal limb muscles in patients who

have experienced anoxaemia or circulatory collapse without evidence of postural pressure.

Most of the recorded cases of muscle necrosis following prolonged coma have been followed by renal failure of varying severity; and it is well to remember that the renal lesion is potentially reversible, although the hazards of sudden hyperkalaemia seem less predictable than in tubular necrosis from other causes. It is important to exclude muscle necrosis in patients developing acute renal failure after coma, particularly where it is associated with anoxaemia, or follows carbon monoxide poisoning, and a suspected diagnosis can be confirmed by muscle biopsy. In Cases 2 and 3 other toxic agents were taken, aspirin in Case 2 and possible barbiturates in Case 3. My conclusions are that anoxaemia and circulatory shock with hypotension produced by carbon monoxide may involve the particularly susceptible area of renal blood flow via ischaemia and vasoconstriction and that the whole picture is yet another example of an acute ischaemuric episode resulting in acute renal failure. I suggest that where the circulatory insult is minimal, muscle necrosis sustained during the period of exposure to carbon monoxide may play a significant role, as in the 'crush syndrome.' The exact mechanism whereby oliguria and renal failure occur remains unsolved—tubular obstruction by casts, disruption of the tubular lumen, diminished filtration as a result of hypotension and cortico-medullary shunt have all been advocated. But Oliver's theory of ischaemuric and toxic causes of tubular damage are based on micro-dissection studies and appear to be well founded.

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Dr. Hunt. I think it must have occurred to most of us that this is very much like a polyarteritis nodosa. Was that the opinion you formed and was there any indication of polyarteritis nodosa?

Dr. Bowen. There was no evidence at all of any glomerular lesion or damage to the kidney blood vessels. Reference was made to the absence of lesions in other vessels in the first case. There was no question of any systemic disease.

Prof. Hill. May I ask the time interval of the anuria and time interval of the hypotension.

Dr. Bowen. In the first case a period of 7 hours occurred during which the patient remained unconscious and within 2 days the urine volume was noticed to be reduced. On the 2nd or 3rd day the blood urea was 163 mg.%. In the 2nd case, the man in the cupboard, 2 days after admission the blood urea was 180 mg.%. The third case, again after rather a short time interval, the blood urea was 186 mg.% I was struck particularly in the third case by the apparent sudden rise in blood urea so soon after the initial insult received. The second and third cases had hypotension when admitted to hospital, but it was not known how long this had been present. In the first case the girl's blood pressure was unrecordable for the first 24 hours, although clinically her condition was good. Unfortunately in the first case there was no accurate urine measurement recorded until it was realized that anuria was evident.

Dr. Camps. One of our main problems is that we are having to deal in retrospect with these cases, and as the average clinician does not bother to take a blood specimen, it is not difficult to understand how you get failure to appreciate what is actually happening.

Dr. Teare. It is a lack of observation on the part of the clinician in not taking blood from people for carbon monoxide examination who come into hospital in coma. Only yesterday I had one half of an elderly couple who were found, one dead and one unconscious, in front of an 'Essee' burner and the unconscious half was labelled as cerebral thrombosis in hospital. The old man was an apparent CO poisoning. I asked the Coroner's officer to go and see what happened at the house. He came back with a story that a sister had moved in there and was having great difficulty in trying to get the 'Essee' burner to light. A CO estimation straight away might have saved a third person from this hazard.

Dr. Camps. This is a perfect example for the universities who deprecate the teaching of this subject. In fact, if every clinician appreciated the importance of this material and saved it, we would get somewhere. It is very common to find that stomach washouts have been thrown away and it is a vital part of the actual diagnosis. My experience is just like that of Dr. Teare.

Diseases Likely to be Encountered in West Indian Immigrants

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There are 200,000 immigrants in the U.K., of whom 100,000 are West Indians. It is therefore important to appreciate the West Indian background, the two main aspects of which are:

i. The racial pattern; and

ii. The dietary background of malnutrition.

The racial pattern is very mixed. The few survivors of the Arawak, or Carib, Indians seldom emigrate and are unlikely to be encountered in the U.K. However, the race preponderating in the genetic pattern is that of the Negro, originating in West Africa, and it tends to dominate any mixture which may include Anglo-Saxon, Portuguese or Spanish. After the liberation of the slaves in the early part of the nineteenth century, many East Indians emigrated to the West Indies and to-day, in Trinidad and British Guiana, they may constitute more than half of the populace. During the latter part of the nineteenth century there was an influx of Chinese and, later, immigrants of Mediterranean origin.

This mixed genetic pattern is of great importance and is illustrated by the presence of sickle cell anaemia or trait in about 5–10% of the populace. Histologically, the spleen in the sickle cell crisis typically shows the Malpighian bodies bathed in pools of sickled blood, and these appearances are almost pathognomonic. However, the red cells in sickle cell trait may sickle agonally or at *post mortem* when fixed in formalin and may prove a trap to the inexperienced pathologist who, in such circumstances will tend to diagnose the cause of death as due to sickling, when in fact it was due to some other cause.

Grossly, the spleen in the sickle cell crisis is diffluent, black and shiny, with rather soft, round areas of infarction or haemorrhage. The spleen in the child or the young adult with sickle cell anaemia will be enlarged, but if the patient lives long enough the organ will contract, due to haemosiderosis and organization of infarction, to a very small size, e.g. 0.5 g. in weight. Besides being a haemolytic disease, sickle cell disease produces much of its pathological effect from infarction due to thrombosis. Such

infarctions are found in the spleen, gut (causing mesenteric infarction), kidney (causing cortical necrosis) and also in the brain, although no organ is immune. A more chronic aspect of this is the now well-recognized avascular necrosis of the head of the femur, which simulates Legg-Perthes' disease.

The electrophoretic pattern is typical, with the slow-moving haemoglobin-S compared with the adult or foetal types. In the trait (heterozygote) haemoglobin-S may be associated not only with adult or foetal haemoglobin, but with other haemoglobinopathies, such as haemoglobin-C. The haemolytic aspects of the disease are well-known, including the enlargement of the marrow cavity and the sun-ray appearance of the skull. At autopsy the presence of pigment gall stones should be looked for.

One associated disease with sickle cell disease is that of osteomyelitis, and it would seem that the hyperactive marrow, with subsequent infarction, may be an ideal medium for circulating bacteria, such as the Salmonellae or Staphylococci.

Finally, it cannot be stressed too often that a sickling test should be carried out on the blood of any patient coming from the West Indies, particularly if the patient is to be subjected to an operation or delivery.

With regard to blood diseases, leukaemias and megaloblastic anaemias are common in the West Indian; but as far as Jamaica is concerned, pernicious anaemia is very rare.

With regard to the second factor in the background, i.e. malnutrition, which produces fatty liver disease in the infants, this liver disease may be one of the possible causes of cirrhosis in West Indian adults who arrive in this country. There is some evidence against this statement, but with the present state of our knowledge of the aetiology of cirrhosis, fatty liver, associated with malnutrition, must be kept in mind.

Another liver disease which may be encountered in this country is that of hepatic veno-occlusive disease (VOD), which is probably caused by the Senecio group of alkaloids found in native bush teas. This liver condition is found in this country in cattle and horses, and is classified under the name of ragwort poisoning. It is known that West Indians are importing the native herbs to make bush teas and one or two cases of VOD have been encountered.

The disease presents itself in a patient of good nutrition, with a swollen belly due to ascites and an enlarged liver. Grossly, the liver is not unlike the nutmeg liver of chronic venous congestion and histologically this is shown to be due to a blockage of the centrilobular veins by a swelling of the intima, with subsequent fibrosis. This fibrosis, which may develop into cirrhosis, is centrilobular in situation and may be found in young adults.

Primary liver carcinoma in the West Indian is not common and only 2 cases have been found in about 2,000 autopsies in Jamaica.

With regard to infective granulomata, tuberculosis is very common in the West Indian. This may be met in the form of pulmonary tuberculosis, tuberculous adenitis or as tuberculous ulceration of the skin.

Leprosy may be encountered, and in Jamaica there are about 1,000 active or healed cases in a population of 1½ million people. Fungus diseases should always be considered in cases of skin ulceration, and several cases of chromoblastomycosis have been described in Jamaica.

Syphilis and gonorrhoea are quite common, despite the introduction of penicillin therapy, and several years ago there were about 1,000 cases of syphilis attending the VD Clinic at Kingston, Jamaica. In Jamaica about 30% of patients attending hospital have a positive Kahn reaction. However, some of these cases may be due to yaws, which is another treponemal disease which is similar to syphilis. Yaws is not a killing disease, but causes deformity and scarring, and these two may be encountered in West Indian immigrants.

Scarring is very prevalent in the West Indian Negro and he is said by some to have a fibrous diathesis. This is illustrated by the prevalence of keloid in the Negro and a high incidence of fibromyomata in Negro women. Similarly, osteogenic fibromata (? atypical Paget's disease) of the jaw are not uncommon and may be encountered in dental practice. Running in parallel with the 30% positivity of the serological tests for syphilis in the hospital populace, these latter also show a 30% positivity to the complement fixation test for lympho-granuloma venereum (LGV).

This disease in the male, in the early stages, is easy to detect because of the formation of inguinal buboes, and later a classical watering-can scrotum and perineum. In the female the disease is much harder to detect, for apparently it gives rise to little discomfort as a vaginal infection. Several years later, however, it spreads to the rectum and produces a rectal stricture, which is not uncommonly encountered, and very occasionally gives rise to a squamous cell carcinoma of the rectum.

Cardiovascular-renal disease is very common in the West Indian. Atherosclerosis in the West Indian Negro is very high and is especially noticeable in the young adult. The incidence of coronary artery atheromatosis is also very high, but the incidence of myocardial infarction is low. The cause of this interesting anomaly is under investigation as it poses the question of the relationship between atheromatosis and myocardial infarction.

Hypertension in the West Indian Negro is very prevalent and is also especially noticeable in the young adult. This high incidence of hypertension can be divorced from the high incidence of nephritis. In many cases Type 1 nephritis, particularly in children, has been shown to be associated with a streptococcal infection of the skin. It has commonly been said that streptococcal infections and rheumatic fever are rare in the tropics. This

does not apply to the West Indies, and a West Indian immigrant may have mitral stenosis or other stigmata of rheumatic fever.

Gastric ulceration is as prevalent as in more temperate climes, but perforation is not common. The incidence of gastric carcinoma is not unusual, but in Jamaica carcinoma of the cervix outnumbers all the other carcinomata put together.

The West Indian does not withstand operative trauma very well, and sudden death half to one hour following operation occurs frequently enough to have a medico-legal importance.

Recently it has been shown that the adrenals of West Indians are about half the size of those of Europeans and that the part which appears to be deficient is the cortex. Further, it has also been found that in severe operations, such as gastrectomy, whereas the European has a transient and mild hyperglycaemia, the West Indian has a hyperglycaemia up to 200 mg. per 100 c.c., and this does not fall for some 24 hours or so.

The West Indian, possibly because of his background of malnutrition, has an unstable carbohydrate metabolism. There has been described a 'Jamaican type' of diabetes. In such a case the hyperglycaemia and glycosuria may be controlled with insulin and then later, without any treatment whatsoever, there appears to be no evidence of the previous diabetes.

The general subject of shock and carbohydrate metabolism is very poorly understood in the West Indian, and is worthy of the intensive research which is now being carried out at the University College of the West Indies.

It is unlikely that cases of malaria in West Indians will be encountered in this country. The liver has a typical slaty or purplish appearance, as has the spleen, which has a diffluent pulp. In cerebral malaria, if a piece of brain is crushed between two slides and put under the microscope, the capillaries can be clearly seen by the black pigment which has been deposited. This is a quick and easy test which is almost pathognomonic of cerebral malaria.